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Toward a Geography of Hormones: The Human Sex Ratio at Birth in the United States 1970-1995

Michael C. Meyers
University of Tennessee, Knoxville

To the Graduate Council:

I am submitting herewith a thesis written by Michael C. Meyers entitled "Toward a Geography of Hormones: The Human Sex Ratio at Birth in the United States 1970-1995." I have examined the final electronic copy of this thesis for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Master of Science, with a major in Geography.

Ken Orvis, Major Professor

We have read this thesis and recommend its acceptance:

Shih-Lung Shaw, Anita Drever

Accepted for the Council:
Dixie L. Thompson

Vice Provost and Dean of the Graduate School

(Original signatures are on file with official student records.)

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TOWARD A GEOGRAPHY OF HORMONES: THE HUMAN SEX RATIO AT BIRTH IN THE UNITED STATES 1970–1995

A Thesis
Presented for the
Master of Science Degree
The University of Tennessee, Knoxville

Michael C. Meyers
August 2004

DEDICATION

This thesis is dedicated to my three sons, my four brothers, and my sister.

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ABSTRACT

It has been hypothesized that humans may exert facultative, adaptive control over the sex of their offspring through the action of the endocrine system. No conclusive evidence of this has been found, although varying hormonal levels in parents at the time of conception may partly influence the sex of the child (James 1986, 1987b, 1999). A decline in the human sex ratio at birth (SRB) observed in the U.S. and some other countries has been attributed by some investigators to widespread environmental exposure to endocrine disrupting chemicals.

The many factors hypothesized to influence the SRB make testing this attribution difficult, but one suggestion has been to explore the geographic and temporal pattern of SRB to determine if a sentinel health event signaled by abnormal SRB is present (Davis et al. 1998). This thesis explores the possibilities of geographic analysis of SRB at various scales, focusing on the local geographic scale of the U.S. county to determine whether patterns of explainable variation exist. It tests the basic geographic-patterning assumption, the hypothesis that hormonally mediated influences such as local socioeconomic conditions, adult reproductive sex ratio, urban versus farm environment, and racial composition may influence the SRB, and looks for posited geographic patterning that might be indicative of hormonally active agents working in the human environment.

This set of hypotheses is tested in univariate and multivariate logistic regression models combining complete U.S. individual birth record datasets for 1970, 1980, and 1990 with selected U.S. Census county-level statistics that were chosen to represent hypothesized socio-environmental, hormonally mediated influences. Separate models were constructed for white and black births, and variables of birth order, plurality, and season of birth were included in multivariate models to control for these confounding individual influences on the SRB.

Results show that geographic patterning is strongly evident at the county level and this approach in general works well to elucidate the influence on SRB of these external hormonally mediated factors. SRB in white populations significantly decreased with increases in county urban population proportion in 1980 and 1990 and with increases of

the percentage of families living below the poverty line in 1970 and 1990. The change in odds ratio for white male births was barely detectable, however, and was less than that found for individual characteristics such as birth order and plurality. Black population SRB was not as influenced by external hormonally mediated factors as white SRB, except in 1980. Little clear evidence for the presumed effects of endocrine disruptors was found.

The results support further study of external hormonally mediated influences on the SRB at local geographic scales. In particular, geographic patterning is strongly evident but varies locally in magnitude and sign, and spatially in pattern, between sampling dates. This suggests that not all significant factors are accounted for in this analysis, and that more work needs to be done to weigh the independent influences of individual biological factors and those external factors that might vary with changes in social, economic, and age-distribution conditions. A significant influence of SRB seasonality in the 1970 sample year also suggests that changes in temperature, light, rainfall patterns and other environmental signals that might stimulate hormonal influence of the SRB should be explored.

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GLOSSARY

–2LL: Negative 2 log-likelihood, reported by SPSS as model chi square of the logistic regression model.

ASR: U.S. Census data reporting age, sex, and race characteristics by U.S. county.

Birth order effect: The tendency of children born first or early in a mother's pregnancy to more likely be male than those born later.

CCDB: U.S. Census data published as the City and County Data Book (CCDB) at approximately 10-year intervals (U.S. Census 1972, 1983, 1994).

CSD: County SRB Dataset. Birth record databases compiled for this thesis containing a number of individual birth characteristics of the child, such as sex, season of birth and mother's age, combined with a number of social and demographic characteristic of the mother's county of residence.

Dizygotic twins: Two children born to the same mother from a single gestation period but from fertilizations of two eggs; also known as fraternal or nonidentical twins.

EXP(B): In logistic regression results presented in this thesis, the exponent of the dependent variable coefficient, also termed the "odds ratio," or OR.

Facultative: A broadly used term in biology to define any activity of an organism that is nonobligated, that is, one in which the individual exerts some control of the outcome. Facultative actions may be adaptive (due to natural selection) or nonadaptive.

Fisherian, Fisher's hypothesis of equal sex ratio: An evolutionarily stable sex ratio is equal (50:50) in most species, because populations that produce more of one sex or another will suffer fitness problems in the next generation.

FSH or LH: Gonadotropins, either the follicle-stimulating hormone (FSH) or the luteinizing hormone (LH) produced in the pituitary gland by both men and women.

Homeostasis hypothesis: An adaptive strategy in which parents increase their reproductive success by producing a child of the rarer sex.

James theory of parental hormonal control: W.H. James has inferred from various evidence that changing levels of gonadotropins, testosterone, estrogen and other gonadal hormones in either parent at the time of conception partly determine the sex of the child.

LH: See FSH.

LR: logistic regression.

NCHS: National Center for Health Statistics, the U.S. agency which assembles and publishes national birth statistics.

Parental age effect: The tendency of children born to younger parents to more likely be male than those born to older parents.

Plurality: Multiple children born to the same mother from one gestation period (e.g., twins, triplets).

RASR: The reproductive adult sex ratio. In this thesis, it is expressed as the total number of males aged 15–49 divided by the total population aged 15–49.

SEHS: Socio-environmental hormonal stressor. Local geographic conditions of adult reproductive sex ratio, racial composition, level of poverty, urban density, and other social, cultural, economic or environmental factors that might alter a resident's hormonal conditions in a way collectively detectable by changes in the SRB.

SRB: The sex ratio at birth. In this thesis it is expressed as the male proportion of live births divided by the total number of births.

SRC: The sex ratio at conception. In this thesis is it expressed as the male proportion of eggs fertilized by Y-chromosome-bearing sperm, divided by the total number of fertilizations.

Trivers-Willard hypothesis, effect: An adaptive strategy that dictates that mothers in good condition will maximize their success in the next generation by producing boys, while mothers in poor condition will maximize their success by producing girls. Devised originally to explain sex allocation strategies in polygynous species where males compete with other males to mate with multiple females (e.g., red deer), it has been used by some authors to explain SRB variation in humans.

Wald statistic: In logistic regression results presented in this thesis, the square of the independent variable coefficient divided by the standard error of that coefficient. It can be interpreted as a measure of the importance of the coefficient's contribution to the model, with higher values representing greater importance.

Z-statistic: In this thesis the one-sample proportion method is used to standardize SRB values among counties with large and small numbers of births for spatial mapping. The difference of the geographic area SRB and the U.S. SRB is divided by the standard error of the population proportion.

Chapter 1. Overview: The Geography of the Human Sex Ratio at Birth

My interest in the human sex ratio at birth statistic began with my long study of and concern with the impacts of environmental toxins on human health, which is also the focus of most of my career as an environmental consultant. Beginning in the 1940s the post-World War II explosion of industrial growth in developed countries produced large quantities of new organic compounds, some of which are still circulating in the environment decades after their last date of manufacture. During the 1970s and 1980s, concern about chemical releases to the environment led to identification of acute and chronic toxic effects to body systems and cancer from exposure to doses containing, in some cases, as little as one part per billion. The common metal-cleaning solvent tricholoroethylene, for example, has contaminated over 830 Superfund sites investigated by the U.S. Environmental Protection Agency (EPA) because concentrations of 1 part per billion or more in groundwater may cause nervous system effects, liver and lung damage, and, in some cases, death (ATSDR 2003). In the 1990s, some researchers (Jensen et al. 1995, Swan et al. 1997, Davis et al. 1998, among others) identified a potential additional category of impact from certain of these compounds, termed endocrine disruptors. Because dioxins, polychlorinated biphenyls, and certain other manufactured compounds are similar in chemical structure in certain ways to human reproductive hormones, they were suspected of disrupting the processes of the human reproductive system by mimicking, blocking, or misdirecting the action of natural hormones. Exposure to doses as small as one part per trillion were capable of such disruption, according to these theories, portending the need for an increase by orders of magnitude of the sensitivity of the regulatory control required to keep these compounds from the environment.

Proof of a disruption of the human endocrine system in any human population has eluded researchers, although highly publicized studies that report seemingly pandemic impacts to human reproductive health suggest a pervasive environmental cause may exist. Some of these studies have sought to detect geographic variation in reproductive system disruptions. Fisch and Goluboff (1996), for example, found significant differences

comparing sperm concentrations in vasectomy patients in Los Angeles, Minneapolis, and New York City, with much lower concentrations found in Los Angeles. What social or environmental factor was operating to cause these differences?

Sperm count concentration studies are criticized because of the differences in how laboratories count sperm and in how sample populations are selected, so a clear picture of geographic variation in U.S. male reproductive health has not yet developed from this work. As I sought a thesis topic that could employ geographic analysis to detect possible impacts from environmental endocrine disruptors, I was pleased to discover two studies from the mid-1990s that suggested that these agents were the possible cause of a change in the human sex ratio at birth observed in many industrial countries. The virtue of the human sex ratio at birth statistic is its remarkable stability, with slightly more males than females born in almost every population measured. The sex ratio at birth is usually expressed as the ratio of males to females born. Beginning in the 1990s, a decline in this ratio—a slight reduction or decrease in the ratio of males to females born—was observed in several developed nations. In the Journal of the American Medical Association, Davis et al. (1998) concluded their alarm over changes in the human sex ratio in these countries by calling for more specific geographic analysis: "Patterns of reduced sex ratio need to be carefully assessed to determine whether they are occurring more generally, whether temporal or spatial variations are evident, and whether they constitute a sentinel health event."

A sentinel health event is recognized as any unusual pattern of disease, disability or mortality that warns of the need for preventative or therapeutic medical care changes at the societal level. Disruption of the human endocrine system by environmental toxins would be a sentinel health event of major proportions, requiring substantial intervention by health and environmental authorities. In 1999 the National Research Council (NRC) Committee on Life Sciences reported that endocrine disruptors in the environment had probably contributed to the declines in some wildlife species and suggested that they may play a role in recently reported declines in human health (NRC 1999). Among weaknesses of past studies, NRC observed that "aggregation of data over larger geographic regions might not be an appropriate spatial scale for this analysis, given the

significant geographic heterogeneity." NRC has identified geographic analysis as a key approach in the research, testing, and monitoring needed to resolve the nature of this environmental threat. In this thesis, I describe the basis for scientific interest in recently noted changes in the human sex ratio at birth, a potential indicator for a profoundly important sentinel health event, and set forth a geographic study that may help determine whether changes in the human sex ratio at birth can be attributed to environmental factors.

The sex of a newborn is attractive as a dependent variable in statistical analysis because it is largely an unambiguous characteristic. Also, statistics for the sex ratio at birth are now recorded for virtually every birth in the United States, and the birth records of many other nations are similarly complete. These features make the live-birth sex ratio potentially useful as a sentinel health indicator, since changes or deviant patterns may alert public health care administrators to emergent problems so that they can begin to investigate and mitigate the avoidable underlying causes. Currently the most commonly used sentinel health indicator is infant mortality, but this statistic is becoming less useful with widespread improvements in obstetrical care. As the underlying mechanism of the sex ratio at birth is illuminated, it may serve better as a baseline measurement of the health of a population, as well as of changes in social or economic conditions.

Studies of the human sex ratio at birth begin with the fact that in virtually all large populations studied, approximately 104 to 107 boys are born for every 100 girls (Chahnazarian 1988). This statistic is variously termed the sex ratio at birth, the live-birth sex ratio, the secondary sex ratio (the primary sex ratio being at conception), or the offspring sex ratio. For the purposes of this study, I will term the live-birth human sex ratio statistic as the sex ratio at birth (SRB). The SRB is also quantified in different ways in the literature. The ratio of boys to girls born in the U.S. is reported by the Journal of the American Medical Association as a simple ratio, so that the 2002 U.S SRB would be 1.05:1. The U.S. Central Intelligence Agency (CIA) records the SRB of every nation as the number of males born for each 100 girls born, reporting the 2002 U.S. SRB as 105. The U.S. National Center for Health Statistics (NCHS) describes SRB as the number of

male births per 1,000 female births, so the U.S. SRB in 2002 was reported as 1,048 (Hamilton et al. 2003). Although not strictly a ratio, the male proportion of births is frequently used in SRB literature instead of the ratio of males to females born (Nicolich et al. 2000, James 2000, and others). So, the male proportion of the ratio of 105 boys to 100 girls in a population is 105/(105+100) = 0.512, or, put another way, 51.2 percent of all births were boys. Because the male proportion convention appears to have been slightly favored in more recent literature, I will adopt it as my preferred expression of the SRB.

The reason for the slight excess of males at birth is not known, but may be the result of natural selection processes (Fisher 1930, 1958) that compensate for the greater vulnerability of the male to accident or disease as he is inseminated, gestates, is born, and grows to reproductive age. While this excess of males is a virtual biological constant, the SRB fluctuates slowly and nonrandomly over time and by place (Gini 1955). Discovering the reason for these fluctuations is a favored pursuit of science, with geographers joining biologists, geneticists, ecologists, anthropologists, sociologists, and physicians trying to discover the central formula of the variation. Darwin (1871) tried an explanation but failed, concluding with this famous challenge: "...I now see the whole problem as so intricate that it is safer to leave its solution for the future."

It is prudent to say that the problem remains intricate and unsolved, although there is a vast literature purporting to explicate the SRB. My initial interest in the influences of environmental toxins on the SRB has grown to a curiosity about the fundamental biological processes that influence it, and appreciation for its profound and immediate application to human ethical and social issues. Nations that suffer a serious imbalance in their reproductive adult sex ratio face social instability. In China and some other Asian countries, the SRB is now at or above 109 (CIA 2003), significantly above the world norm. The surfeit of males in these countries in another decade looms as a potential destabilizer of global security, as social instability translates to bellicose foreign policy. At same time, the sex ratio of a family is one of its most defining but unpredictable characteristics and influences such fundamental decisions as how many children a couple will have. In this thesis I hope to show that the study of the human SRB has worthy

benefits and that geographic analysis will contribute to a better understanding of its nature.

The world geography of the SRB

Determining the worldwide pattern of SRB is problematic because as many as 50 million births per year may be unregistered worldwide, approximately 40 percent of all births (UNICEF 2002). However, the CIA provides best-estimate values for 222 nations (CIA, 2003). In 2002, the mode of national SRBs was .512 [105], with the U.S. and 168 other nations reporting that value. The mean SRB of all CIA-monitored nations in 2002 was .511. Only four countries, Faroe Islands, Grenada, Bermuda, and the Cayman Islands, have an SRB of .500 or less. Figure 1 displays CIA-estimated national SRB values for 2002. Deviations from the modal SRB are worth examining, because they illustrate some factors that may mitigate or confound environmental influences on the SRB.

The highest SRBs

National SRB values may reflect cultural factors favoring boys or girls. For example, population researchers have observed that where a strong preference for sons exists in conjunction with a low birth rate, the sex ratio will be very high. At .522 [109] in 2002, China's SRB is among the highest in the world (CIA 2003) and may have reached as high as .537 [116] in the 1990s (Tuljapurkar 1995). Taiwan, South Korea, Singapore, and a few other countries have similarly high SRBs (CIA 2003). India's SRB was as high as .519 [108] in the late 1990s and has been reported to be very high in some western and southern states (Retherford and Roy 2003). During the 1960s and 1970s, the SRB in China was closer to the world mean value but has steadily increased since the 1980s (Gu and Li 1994). South Korea, Taiwan, and India show similar trends (Posten et al. 1997). In these countries, the SRB values for first children are similar to that of other nations, but climb much higher for second and later children as parents intervene in pregnancies to exert their preferences for boys. Ultrasound machines are mass produced and widely available throughout these countries and elective abortion is believed to be

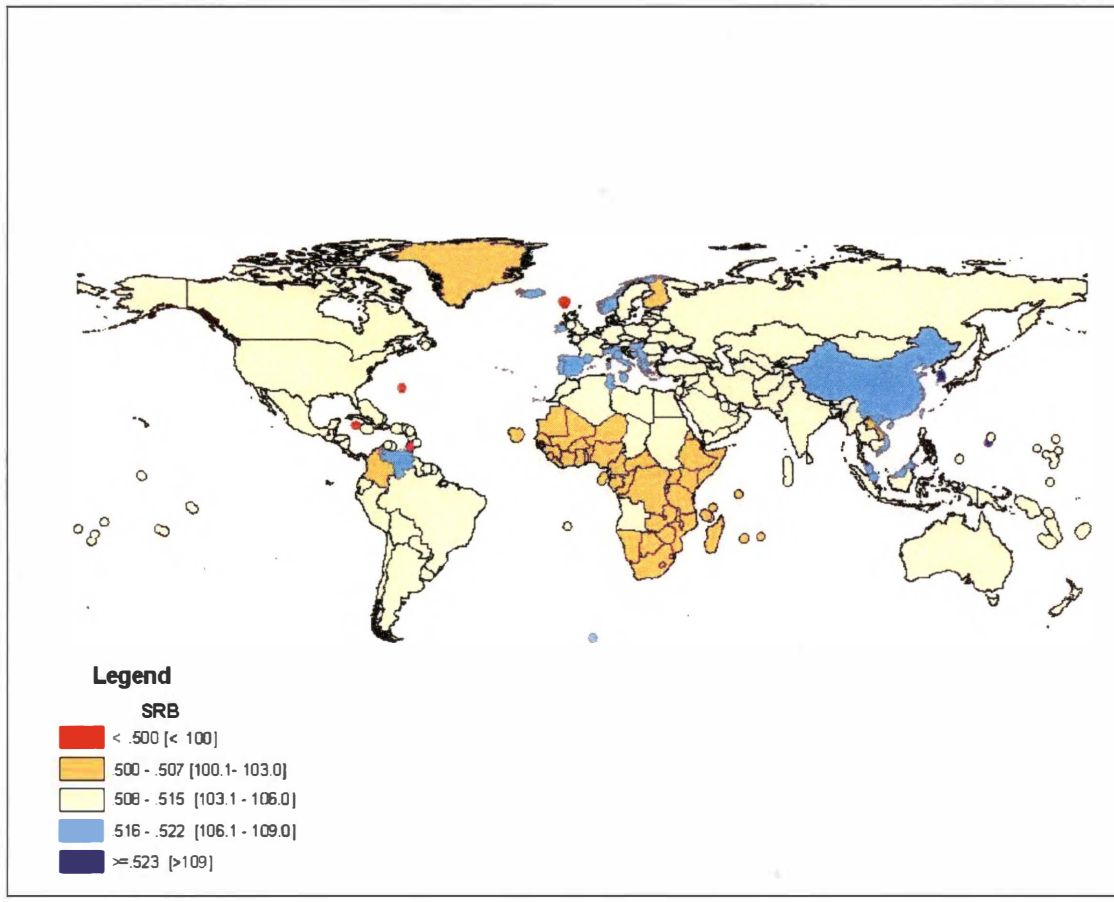


Figure 1. National SRB values in 2002 (island nations buffered for visibility) (CIA 2003)

significant. In China, the state enforcement of family planning since 1978 is a factor, but the strong Confucian preference for sons combined with voluntary reductions in family size in South Korea and Taiwan may also increase the male proportion of births. In India, Bangladesh and Morocco, large bride dowries impose a financial burden on parents of daughters that may contribute to the excessively high SRB recorded there. In 1996, India banned the use of ultrasound machines for determining the sex of a child, but the practice persists and SRB remains very high, especially in rural provinces (NYT, October 27, 2003).

The lowest SRBs

Compared to the SRB of U.S. whites, a lower SRB among black populations in the U.S. has been recorded for at least a century (Winston 1931). Many of the lowest national SRBs reported globally are in nations on the African continent where a ratio of less than .510 [104] is common (CIA 2003). Registration records in Africa are generally poor, however, and there is debate about the true SRB in these countries. The lowest SRBs in Africa are in countries with predominantly black populations and are similar to those reported for U.S. blacks. Some researchers charge that demographers have simply applied the U.S. black SRB value to African countries in the absence of a good system of vital statistics in these countries; some report higher SRBs among black African populations than those typically reported. Ayeni (1975) calculated an SRB of .514 among the Yorubas of southwestern Nigeria during a seven year study, near that of European and North American whites. Rehan (1982) found an SRB of .517 [107] in a review of hospital records delivering Hausa children in the Katsina province of Nigeria born between 1961 and 1980.

Garenne (2002), however, found that the average SRB in 56 demographic surveys conducted in African populations was .508 [103.3], with distinct SRBs in three geographic and ethnic subsets. One subset had SRBs like those usually cited for African populations (.509 [103.5]); another subset of high SRBs (.517 [107]) was found in a few countries such as Nigeria and Ethiopia, and one subset had lower sex ratio, particularly among Bantu populations in eastern and southern Africa (.502 [101]). While the matter

of biased SRB estimates in African countries remains unresolved, the preponderance of current information suggests that there are unknown factors that make the black SRB lower than white populations worldwide.

Late 20th century SRB declines in industrialized nations

SRBs in industrialized countries are generally about .512 among white populations (CIA 2003). However, a trend of decline in the SRB in several industrialized countries has been noted by many authors in studies completed during the last decade. These trends of decline have been found in the U.S., Europe, Latin America, and Japan, beginning, in the opinion of some of these researchers, with the onset of widespread industrialization and the release of chemicals from industrial processes into the environment (Allen et al. 1997, Davis et al. 1998, Jongbloet et al. 2001, and others). Declines in the SRB of Danish newborns have been recorded beginning in 1950, after a rise over the previous century (Moller 1996). Allan et al. (1997) found that the SRB decreased approximately .0022 in the Canadian population from 1970 through 1990. A similar decline of .001 was found in the U.S. during the same period (Allen et al. 1997). As summarized by Devra Lee Davis of the World Resources Institute and her colleagues in the Journal of the American Medical Association (Davis et al. 1998), each of these studies have recorded small but significant declines beginning in 1950, 1960 or 1970 (Table 1).

Declines have also been observed in Sweden, Germany, Finland, England, and Wales (Dickenson and Parker 1996). Not all studies of national SRB trends have revealed consistent declines, however, and the simple association of industrialization and SRB decline breaks down under close scrutiny. The starting period of decline differs from study to study. Uchida et al. (2000) found the Japanese SRB increased from the period 1925 to about 1960 and then decreased thereafter. Was this decline attributable to later industrialization in Japan relative to Europe, or some other factor? Two broad studies of national trends motivated by concerns about SRB declines have somewhat contradictory results. Parazzini et al. (1998) analyzed trends between 1950 and 1990 in 29 countries from five continents and found that SRB had not declined in most countries, but rather had remained constant. They did note, however, decreasing trends in some

Table 1. SRB Declines in Industrialized Countries

Country	Years	Difference in SRB (Male Proportion)	p Value
Canada	1970–1990	–0.0022	<.001
United States	1970–1990	–0.001	<.001
Denmark	1960–1995	–0.002	<.010
The Netherlands	1950–1994	–0.003	<.001

northern and eastern European countries, Greece, Portugal, and, particularly, Mexico, but found increasing trends in southern Europe and Australia. Martuzzi et al. (2001), on the other hand, found a significant linearly decreasing trend in annual SRB of approximately .0001 [10 births per 100,000] in 23 European countries between 1950 and 1986. In eighteen of these countries SRB was lower in the 1993–1996 period than in the 1950–1953 period. However, the SRB increased in some countries and, as did Parazzini et al. (1998), they found regional and national differences in the trend. These variations in SRB trends among countries indicate that sociodemographic characteristics might explain trend differences, rather than a pervasive exposure to environmental chemicals from industrial processes or agricultural activities. The World Health Organization found that SRB varied by latitude in Europe and North America, with more boys born in southern latitudes in Europe and in northern latitudes in North America, indicating that genetic and environmental differences may also be a factor in SRB variation (Grech et al. 2002a).

Higher SRBs in Italy and Ireland may illustrate the operation of one potential sociodemographic factor. The SRB in Italy has increased from one of the lowest in Europe to one of the highest (.519, CIA 2003). Ireland's is also similarly high at .517. Perhaps as Ulizzi and Zonta (1995) suggest, birth control in predominantly Roman Catholic Italy (and, I would also speculate, Ireland) has become widespread later than in the rest of Europe, and has resulted in a significant and relatively recent decline in family size. As more boys are born in earlier than in later births, a decrease in national median family size might increase the SRB.

Reports of late 20th century declines in industrialized countries beg the question of how SRB has varied over longer periods of time. Based on limited information, it appears that the slight excess of males is a historical trend of long standing, although the precision of estimates is in question. Graunt (1662) may have reported the first SRB statistic, calculating a male proportion of .515 to .516 in the infants christened in 17th century London churches. The British national registration system began in 1841, but most national birth registration records do not begin before the 20th century. Motivated by tax collection or military conscription, early national registrations considered females as economic dependents of their fathers or husbands and generally over-report male

proportion (Chambliss 1949). Church birth and christening records may be less biased and precede the institution of national vital statistic reporting. Using church parish records, Vartiainen et al. (1999) found the SRB trend in Finland increased during the period 1751 to 1920, and then decreased thereafter, interrupted by peaks during and after World War I and World War II. These trends were not explainable by individual parameters believed to influence the SRB, such as birth order or parental age. The peaks during wartime reported here have also been observed in other populations, but the overall decline in SRB in Finland occurred before the period of industrialization that earlier studies have associated with some national declines.

U.S. SRB trends

In the U.S., birth statistic reporting is the responsibility of local, usually county- or state-level, health agencies. Historically, the U.S. Census Bureau has collected natality and mortality statistics from local agencies. The U.S. birth registration system began in 1915 with 10 states and the District of Columbia. Prior to 1933, when all 48 states began participation, a national geography of U.S. births can only be estimated. Winston (1931) estimated SRB in the U.S. from 1915 to 1927 at a mean of .514 [105.8]. Examining the SRB for racial differences, already well known by the time of his study, he found that rural whites had a slightly higher SRB than urban whites (.5150 [106.2] and .5148 [106.1], respectively). Blacks had a lower SRB than whites, but those in rural areas also bore more boys than those in urban populations (.510 [104] and .506 [102.4], respectively). Higher rural SRB and higher SRB in whites compared to blacks were frequently noted characteristics in early U.S. SRB studies.

Urban-rural distinctions are common in SRB studies, but few researchers have closely scrutinized other geographic distinctions. Chambliss (1949) was one of the first geographers who undertook to analyze the SRB in detail in the U.S. Studying the SRB for all U.S. states for the period 1915 to 1944, he found that the SRB for native whites was .515 [106], somewhat higher than the SRB for foreign born and native whites combined (.514 [105.8]). He excluded black births from his study because "...colored groups in the United States have uniformly, in large samples, a lower sex ratio than white groups." He found significant differences among U.S. states, with Kentucky and North

Carolina having consistently high SRBs, and Louisiana and South Dakota consistently low ones. But he concluded that the striking differences among states were due to clerical errors and differences in the completeness of registration in these states, not geography. The U.S. SRB, he concluded, had probably gradually risen through the century but this rise was obscured by improvement in registration of female births. He found that states with a superior birth registration had lower SRBs than those with poor registration. He also speculated that the differences in SRBs among races has something to do with reporting differences, noting that the SRB of blacks had not increased over the course of the century even though it was probable that their health care and economic condition, factors then considered possible influences on SRB, had improved.

Chambliss' work provides two cautionary guides for geographic research into the U.S. SRB: racial distinctions and reporting systems that differ by state. The recent decline in the U.S. SRB was first noted by Allen et al. (1997), although their study concentrated on the more significant declines in the Canadian SRB. The Canadian SRB, they found, had declined since 1970 in an east to west gradient, with the greatest decline in the Atlantic region (–.0056; $p<0.01$). They did not find a similar east-to-west gradient in U.S. regional divisions (defined in Table 2), although they found significant differences among these regions. Marcus et al. (1998) confirmed that the U.S. SRB had indeed declined significantly, from .513 [105.3] in 1969 to .512 [104.9] in 1995, but made a distinction that Allen et al. had overlooked: the U.S. decline is confined to births among *white* mothers.

During the same period the SRB has significantly *increased* among black newborns (Marcus et al. 1998). Within each of the nine geographic regions of the U.S., the white birth ratio declined and the black birth ratio increased during this period except for a decrease in black SRB in the Pacific region. The different trends in these two racial groups weakens the theory of pervasive environmental exposure offered by Allen et al. (1997) and Davis et al. (1998). However, there seems little dispute that there is a longstanding difference in SRB between black and white populations in the U.S, and it appears that SRB is currently trending differently in these groups. In a review of the secular changes in SRB in 41 primarily national populations over the last 50 years, James

Table 2. U.S. Census Bureau Regional Divisions and the States They Comprise

Division	States
New England	Maine, New Hampshire, Vermont, Massachusetts, Rhode Island, Connecticut
Middle Atlantic	New York, New Jersey, Pennsylvania
East North Central	Ohio, Indiana, Illinois, Michigan, Wisconsin
West North Central	Minnesota, Iowa, Missouri, North Dakota, South Dakota, Nebraska, Kansas
South Atlantic	Delaware, Maryland, District of Columbia, Virginia, West Virginia, North Carolina, South Carolina, Georgia, Florida
East South Central	Kentucky, Tennessee, Alabama, Mississippi
West South Central	Arkansas, Louisiana, Oklahoma, Texas
Mountain	Montana, Idaho, Wyoming, Colorado, New Mexico, Arizona, Utah, Nevada
Pacific	Washington, Oregon, California, Alaska, Hawaii

(2000) found significant changes only in three populations: a decrease of white SRB in the U.S., an increase in white SRB in Australia, and an increase in black SRB in the U.S.

NCHS dataset for the study period

This chapter concludes with a geographic portrait of the U.S. SRB decline and a description of the national birth registration dataset that I will use to test my general hypothesis that geographic analysis of SRB may reveal something of the nature of the factors that influence it. The period of decline in national SRB appears to vary by country, but 1970 marks a period at or near the beginning of the U.S. decline. I considered birth registration adequacy and made some attempt to evaluate historic trends prior to 1970. In response to cautions in the literature to be sure to consider demographic factors, I also distinguished between SRB in white and black populations during this period.

In the early 1960s, the National Center for Health Statistics (NCHS) assumed primary responsibility for natality statistics collection. In addition to publishing its own research, the NCHS makes primary statistics available to the public in a number of forms. The most detailed is the Natality Data Set, an annual record of several demographic and health indicators extracted from each birth certificate recorded in the U.S. These data are collected from 50 states and the District of Columbia. To avoid disclosure of the personal identity of a particular record, the NCHS geographically aggregates, in its published datasets, data for cities and counties with smaller populations. For example, the data fields for county of occurrence (where the birth took place) and county of residence (where the mother lives) are specific only for counties with populations greater than 100,000 in the year of reporting. Births occurring in counties with smaller populations are reported as "other county" for that state. While this threshold identifies the county location of approximately 75 percent of the total U.S. births in recent years, most of the land area of the U.S. is excluded, including many of the rural and agricultural areas important to chemical exposure analyses in the study I anticipated undertaking.

For the purposes of this study, I requested and received from NCHS a special version of the Natality Data Set file that includes all fields and records released in the public datasets and, additionally, the county of residence and county of occurrence fields

for every birth. I received such a complete dataset from NCHS for 1970, 1975, 1980, 1985, 1990, and 1995, spanning the study period. These data are provided in ASCII format accompanied by documentation of field structures and other characteristics of the dataset. These data were used for all analyses reported in this thesis and, per my agreement with the NCHS, no individuals were identified using these data. Hereafter I will refer to these data collectively as the study period dataset.

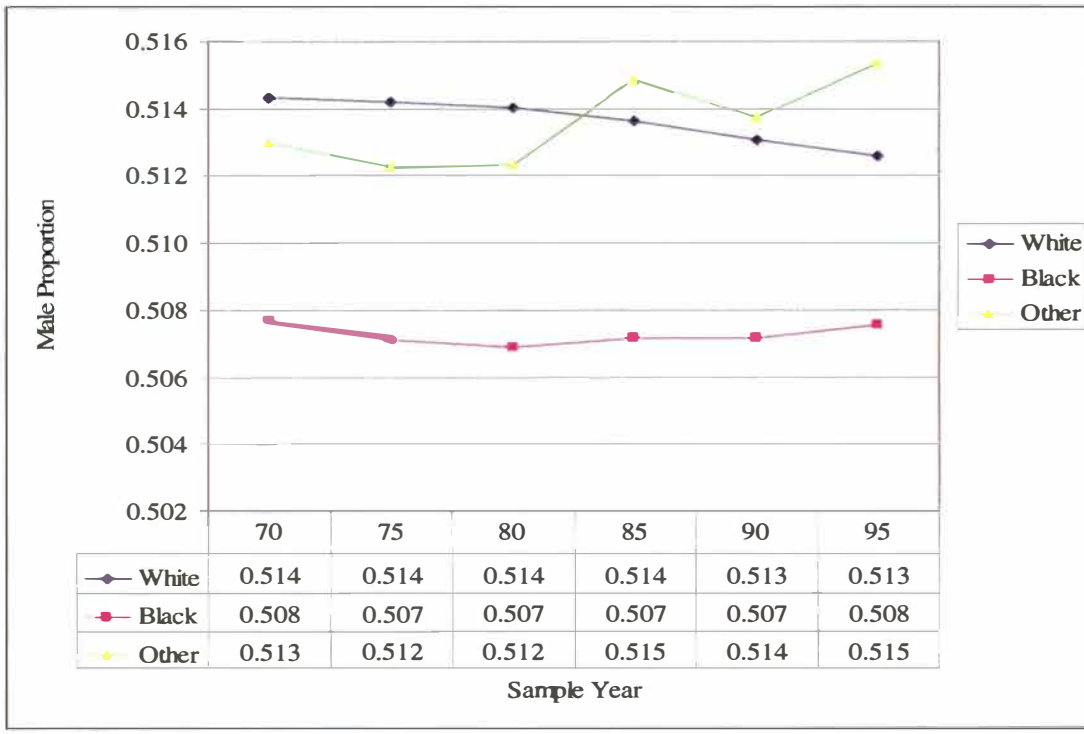
Although the geographic area of the study period dataset includes 50 states and the District of Columbia, a complete 100 percent sample of each birth is not available until the 1985 data year. In previous years, certain states provided 50 percent sample datasets; in 1970, the entire dataset compiled and released by NCHS is based on a 50 percent sample. Table 3 shows the number of records in each annual dataset and the number of U.S. resident births recorded for each year. Births to residents of foreign countries are recorded in geographic fields of place of occurrence but are not included in any U.S. place-of-residence counts.

In the studies cited above and later in this thesis, the separation of SRB values by racial category is frequently used to isolate this factor from other suspected influences on the SRB, recognizing the consensus that this factor has consistently biased SRB. To compare the SRB trends reported above with those in the study period dataset, I plotted for each year the SRB values for total U.S. births and for U.S. births by three racial codings: white, black, and all other races (Figure 2). Although not measured for significance, the decline in the total SRB for the study period dataset shown on this figure confirms the observations of Allen et al. (1997) and Marcus et al. (1998) of a total decline in the U.S. population SRB. It also confirms the observations by Marcus et al. (1998) that the decline appears to be confined to white births. The significant increase in black birth SRB reported by Marcus et al. (1998) is not apparent in this plot. However, I have assembled birth statistics from NCHS for the period from 1940 to 2001 that show that the gap between the SRB for whites and blacks has considerably narrowed during this period (Figure 3).

I defer to Marcus et al. (1998) and others who have reported the SRB decline in the U.S. population and do not independently attempt to confirm the significance of this

Table 3. Number of Births in Study Period Dataset vs. Total U.S. Births

Year	NCHS Dataset	Total U.S. Births
1970	1,865,693	3,731,386
1975	2,232,406	3,144,198
1980	3,310,301	3,612,258
1985	3,760,561	3,760,561
1990	4,158,212	4,158,212
1995	3,899,589	3,899,589
Total	19,226,762	22,306,204



	70	75	80	85	90	95
White	0.514	0.514	0.514	0.514	0.513	0.513
Black	0.508	0.507	0.507	0.507	0.507	0.508
Other	0.513	0.512	0.512	0.515	0.514	0.515

Figure 2. U.S. SRB (male proportion) by race in study period dataset (NCHS 2003b)

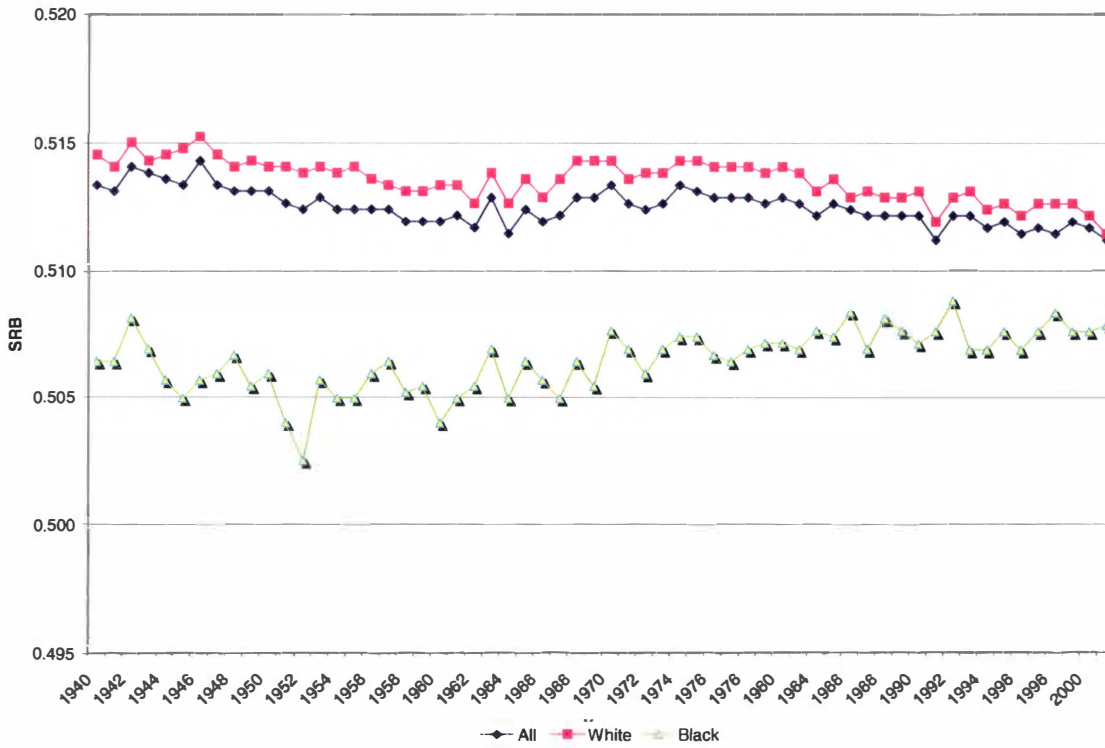


Figure 3. U.S. SRB by year, births to total population, white population, and black population, 1940–2001 (NCHS 2003b)

decline in my smaller dataset. Because these declining trends are also present in my study dataset, I assume this sample is useful for beginning a study of the geographic distribution of the U.S. SRB decline. The trends in white population SRB decline in the study period dataset seem to increase markedly after 1980, compared to the two earlier intervals. Whether this acceleration is an effect of the increasingly comprehensive nature of the dataset or an indication of the timing of the decline is worth considering in subsequent analyses.

I have found no study since Chambliss (1949) that examines SRB within geographic areas smaller than the U.S. census division. Chambliss' attempt to study U.S. SRB was constrained by incomplete birth registration, as discussed above, but the improvements in U.S. birth registration practices merit a return to his perspective. The study period dataset does, however, have some limitations that affects its geographic accuracy. I use the NCHS mother's county of residence as the geographic identifier for all analyses in this study. Where this information is missing, NCHS or the state vital statistics agency may substitute the county where the birth occurred, which could be very different than the geographic area where the conception and gestation of the child occurred. NCHS also estimates that births will be overly recorded as urban because of the practice of nonurban residents having children in urban hospitals and also because many rural residents are assigned addresses within the nearest city, because postal addressing requires a city or town identifier. However, the study period dataset appears to have a high level of data quality for geographic analysis. Although all of the 1970 NCHS dataset and approximately 10 percent of the 1980 NCHS dataset is based on a 50-percent sample of recorded births, the sampling area is the county, so at least 50 percent of the births occurring in any U.S. county during any sample year are represented. NCHS does not report the completeness of reporting for the county of residence field, but in the 2002 NCHS dataset a related field, the mother's place of birth, was missing on the birth certificate in less than 0.2 percent of all records.

By the mid-1970s, the completeness of the data is estimated by NCHS to be 99.3 percent. Over the period of my study the error rate from incorrect transcription and coding is expected to be less than 2 percent per item, based on NCHS data quality control

requirements. By 1985, 100 percent of registered births, representing 99 percent of the births in this country, are recorded in the study period dataset, with recording completeness for white births slightly greater than for that for other races.

In 1989, NCHS announced that it had changed its methods for classifying race in reported tabulations. Beginning in 1989, birth data that report racial characteristics use the race of the mother instead of race of the child. Prior to 1989, the race of the child was coded using an algorithm that considered the race of both parents, generally coding the child as nonwhite if either parent were. I use the NCHS race-of-child field in my analyses throughout the study period, so my tabulations based on race for 1990 and later may be somewhat different from those published in NCHS reports.

Spatial mapping of U.S. SRB values

Geographic mapping and analysis of SRB have been attempted by only a few researchers (see, for example, Oberg 1990) and there are no established methodological procedures for such an analysis. In the case at hand, thematic mapping may bring to light spatial patterns that would invite more detailed analysis, provided that the SRB can be standardized to account for varying sample sizes among geographic areas and for year to year fluctuation. My general approach for initial evaluation is to standardize, graph, and map SRB values at the state, regional division, and county level. To standardize SRBs, I adopted the Z-statistic method used by Bohning and Ayutha (1999) in their study of SRB in Thailand. I use the standard one-sample proportion method in which I assume the male proportion of the sex ratio in the U.S. for every given year is λ, where λ is the total number of male births n_M in the United States in that year, divided by the total number of live births n. Let

$$\hat{\lambda}M = n_M/n_i \text{ for } i = 1,\ldots.N. \qquad (1.1)$$

where $\hat{\lambda}M$ is the SRB, or male proportion of total live births in geographic area i. The one-sample z-statistic for the SRB of geographic area i is therefore the difference between the SRB of that area (AM) and the SRB of the U.S. as a whole in that year, divided by the standard error of a population proportion, or:

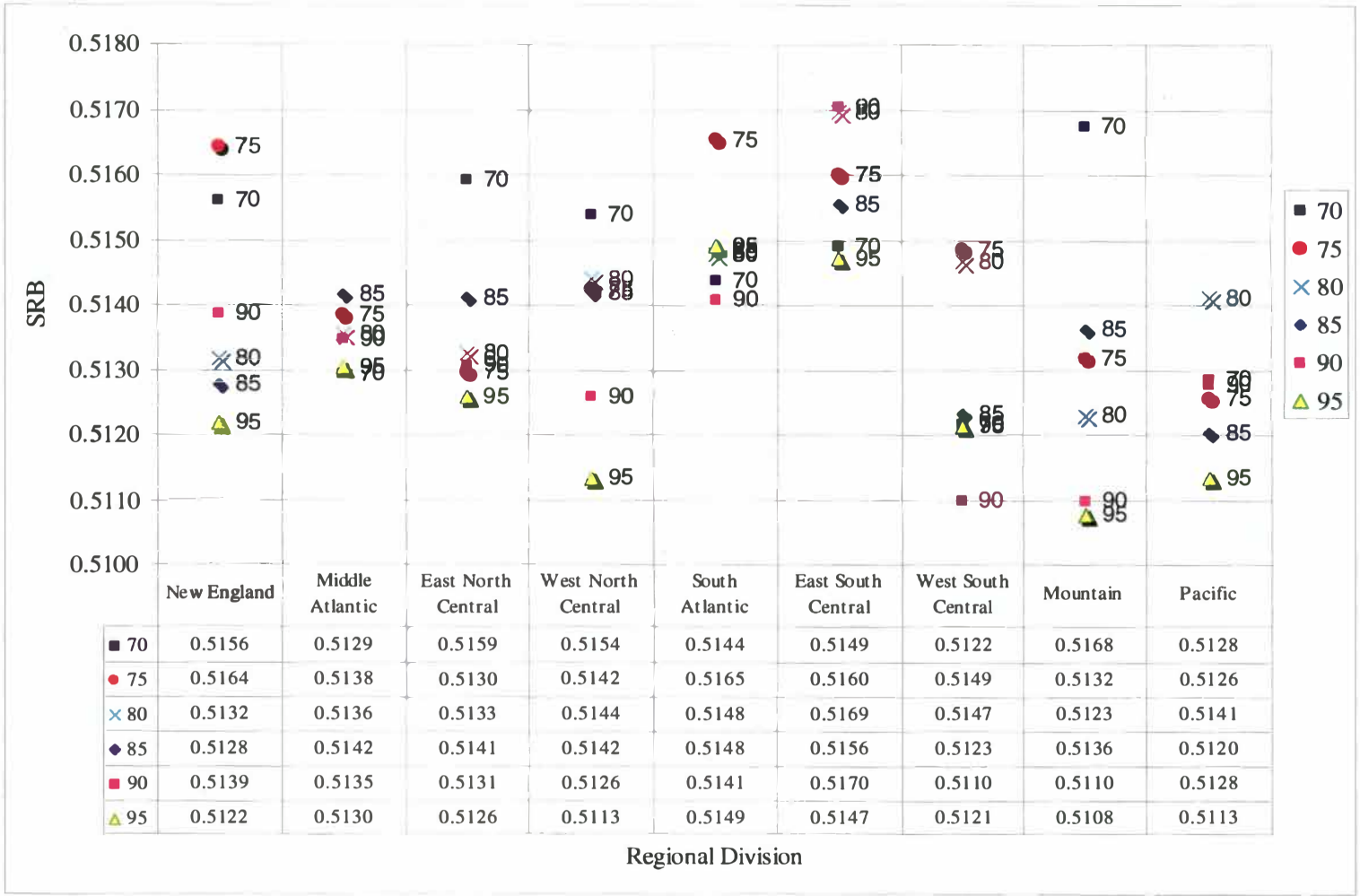
$$Z_i = \frac{\lambda\hat{M} - \lambda}{\sqrt{\lambda(1-\lambda)n_i}} \quad \text{for i = 1,....,N.} \qquad (1.2)$$

This procedure allows us to map areas of significantly low or high SRB, or, in Z-statistic terms, $Z < -1.96$ or > 1.96. Negative values represent significantly high female births; positive values, high male births. The Z-statistic describes how far the SRB in any geographic area is from the U.S. SRB in terms of its standard error. There is an inverse relationship between the standard error of the proportion and the sample size, so geographic areas with a small number of births will have large standard errors. In 1970, for example, both Perry County, Arkansas and Los Angeles California had identical SRBs of .509, and both were less than the U.S. 1970 total SRB of .514 by the same amount. However, the Z-statistic shows that births are significantly lower in Los Angeles ($Z= -2.37$) in 1970 because its large number of births (54,996) yield a smaller error term in the calculation of population proportion, while the 55 births in Perry County yield a large standard error and thus a nonsignificant decrease in SRB ($Z=-0.077$). For these reasons the Z-statistic can be used across sample years to compare the geographic distribution of high and low SRB values, relative to a particular sample year.

While this approach has some flaws—particularly in the case of low birth numbers and extreme proportions—it will serve for descriptive thematic mapping of the spatial variation of SRB in small geographic areas of the U.S. and allow year by year visual comparison of these variations. Evaluation of the consistency, or lack of consistency, in these patterns is important to the question of how a hypothesis of geographic variation in SRB must be formed.

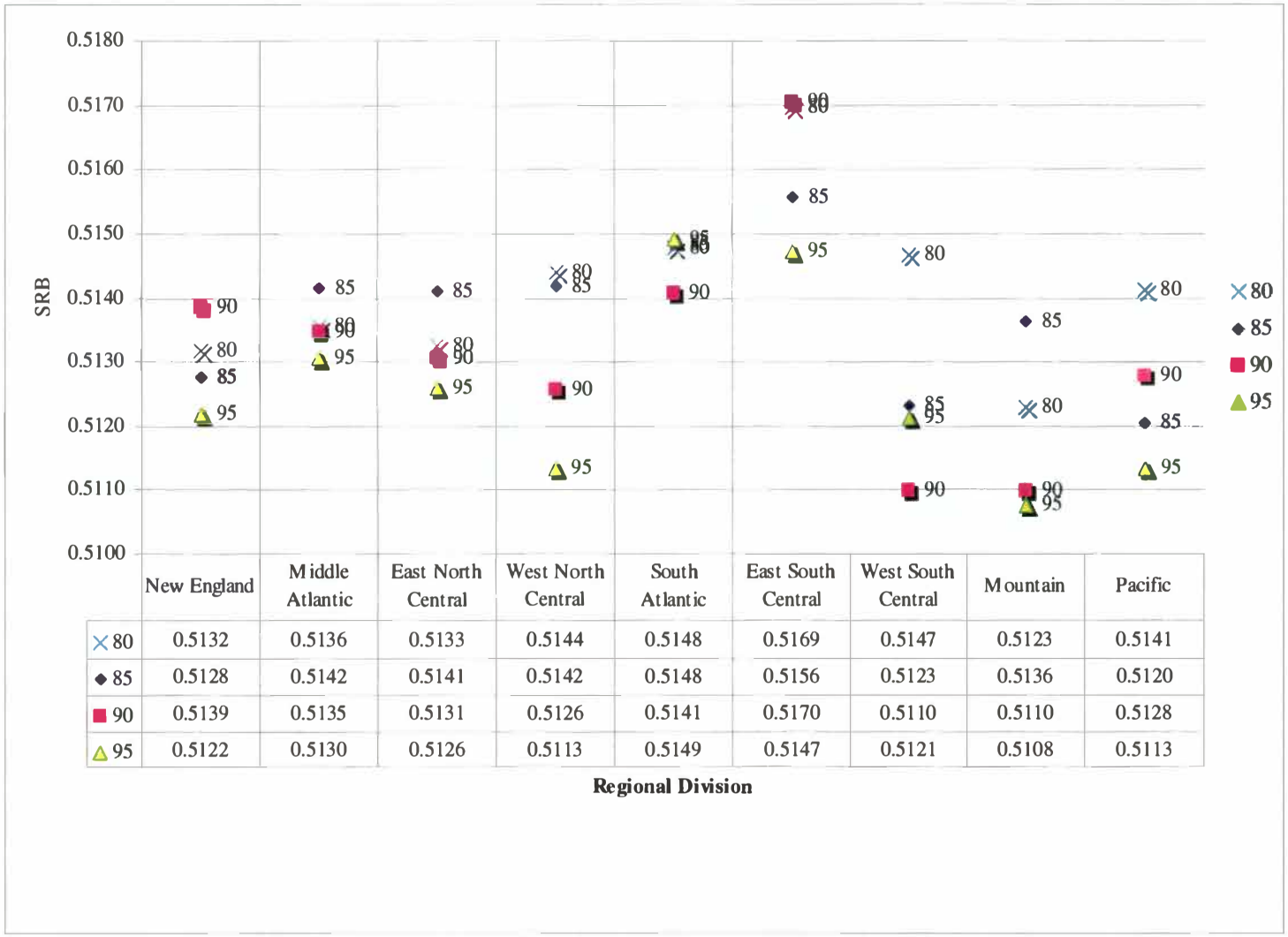
Geographic distribution by U.S. census division and state

Figures 4a and 4b plot the divisional differences in the male proportion of white births in the nine regional census divisions of the U.S. Figure 4a presents the raw male proportion value grouped by regional area for all six sample years; Figure 4b presents the same data for the final four years of the study period dataset. Figure 5 presents the same data by sample year rather than division. These figures show noteworthy regional differences in the SRB. The East South Central and South Atlantic regions have



	New England	Middle Atlantic	East North Central	West North Central	South Atlantic	East South Central	West South Central	Mountain	Pacific
70	0.5156	0.5129	0.5159	0.5154	0.5144	0.5149	0.5122	0.5168	0.5128
75	0.5164	0.5138	0.5130	0.5142	0.5165	0.5160	0.5149	0.5132	0.5126
80	0.5132	0.5136	0.5133	0.5144	0.5148	0.5169	0.5147	0.5123	0.5141
85	0.5128	0.5142	0.5141	0.5142	0.5148	0.5156	0.5123	0.5136	0.5120
90	0.5139	0.5135	0.5131	0.5126	0.5141	0.5170	0.5110	0.5110	0.5128
95	0.5122	0.5130	0.5126	0.5113	0.5149	0.5147	0.5121	0.5108	0.5113

Figure 4a. White SRB (Male Proportion) by division and sample year in study period dataset



	New England	Middle Atlantic	East North Central	West North Central	South Atlantic	East South Central	West South Central	Mountain	Pacific
80	0.5132	0.5136	0.5133	0.5144	0.5148	0.5169	0.5147	0.5123	0.5141
85	0.5128	0.5142	0.5141	0.5142	0.5148	0.5156	0.5123	0.5136	0.5120
90	0.5139	0.5135	0.5131	0.5126	0.5141	0.5170	0.5110	0.5110	0.5128
95	0.5122	0.5130	0.5126	0.5113	0.5149	0.5147	0.5121	0.5108	0.5113

Figure 4b. White SRB (Male Proportion) by division and sample year for 1980, 1985, 1990, and 1995

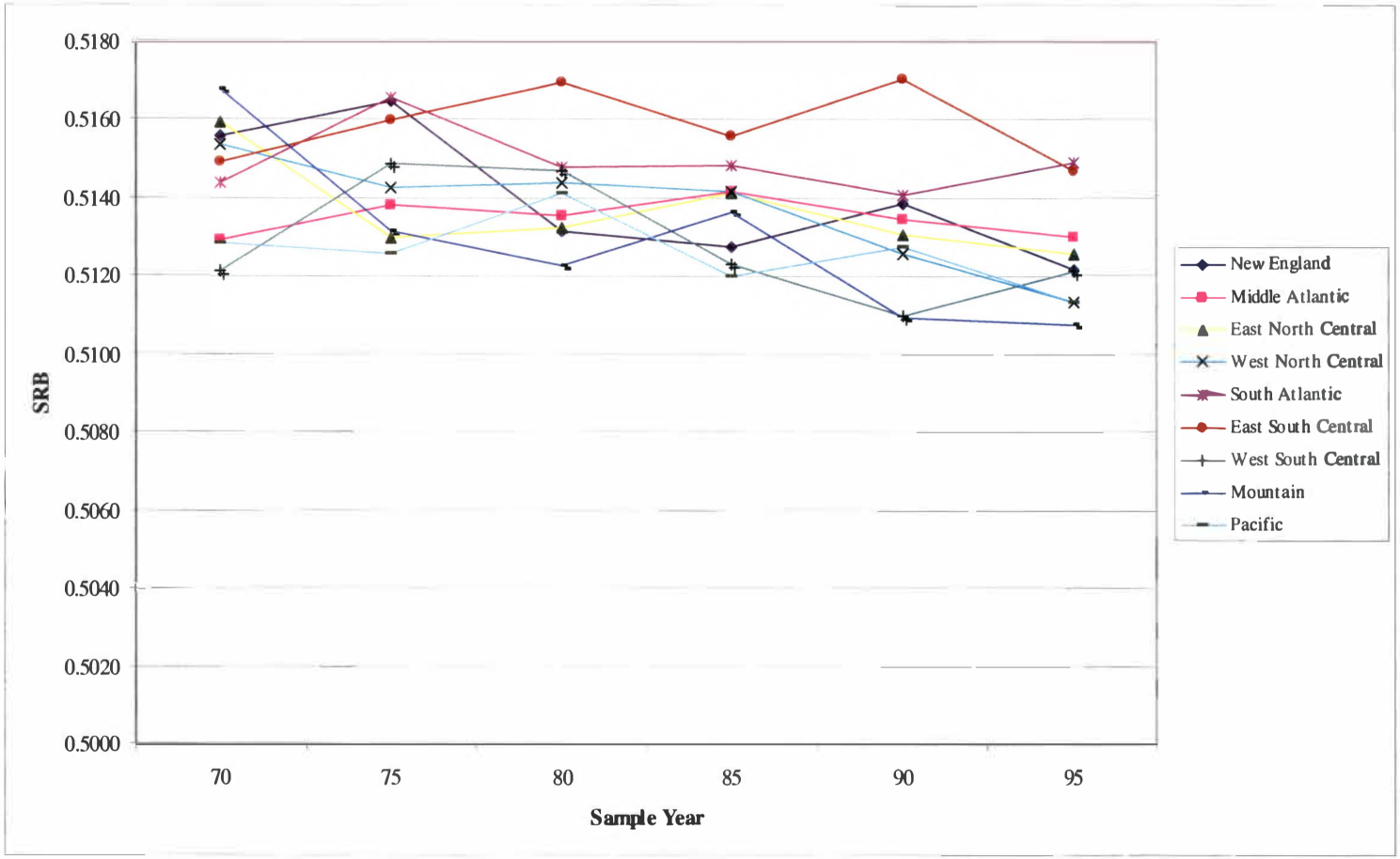


Figure 5. White SRB (Male Proportion) by sample year and division in study period dataset

consistently higher SRBs than do any other regions during the study period, while the Pacific and mountain regions have consistently lower values. However, the decline in SRB appears to affect all regions, although less so for the South Atlantic and East South Central regions. Figure 6 shows divisional differences in black births by sample years. The distinct regional variation present in the white birth dataset is not present for these births.

Thematic mapping of the State SRB during the study period yields additional information about the location and intensity of the decline in U.S. values. Figure 7 thematically plots the male proportion of births for each study year using identical MP value ranges for each year. It is clear that SRB varies considerably year by year within a state, with some states vacillating through the range of SRB values in the course of the study period. States with high SRB values are considerably fewer in number in 1995, but concentrations of high and low values appear more pronounced in 1995. The Midwest and Mountain states appear to have lost SRB value in relation to the South and states bordering the South. Converted to Z-statistics, some concentrations of SRB values are notable. Table 4 lists states that have significantly high or low Z-statistic SRB values for births when totaled for all six of the sample years; these further confirm the higher SRB values for southern states and lower values for western states. The SRB for Texas and New Mexico is significantly lower than the mean U.S. value for the reporting year in two of the six sample years.

Figure 8 plots black births during the same period, but the Z-statistic is used to correct for the low black birth counts in many states. As noted above, declines in black SRB have not been observed, so the Z-statistic plot in Figure 8 shows the relative distribution of state black birth SRB values within each year. The state black birth SRB is not significant (Z statistic <-1.96 or >1.96) for either male or female excess births in any state for one than one year, except in Mississippi (1990: –2.541; 1995: –1.976) and Oklahoma (1980: –3.061; 1985: –2.426) where the SRB is significantly lower in two years, and in West Virginia (1980: 2.499; 1995: 2.001). The regional pattern of trends is not consistent from year to year. Table 5 lists states that have significantly high or low Z-statistic SRB values for black births when totaled for all six of the sample years. No

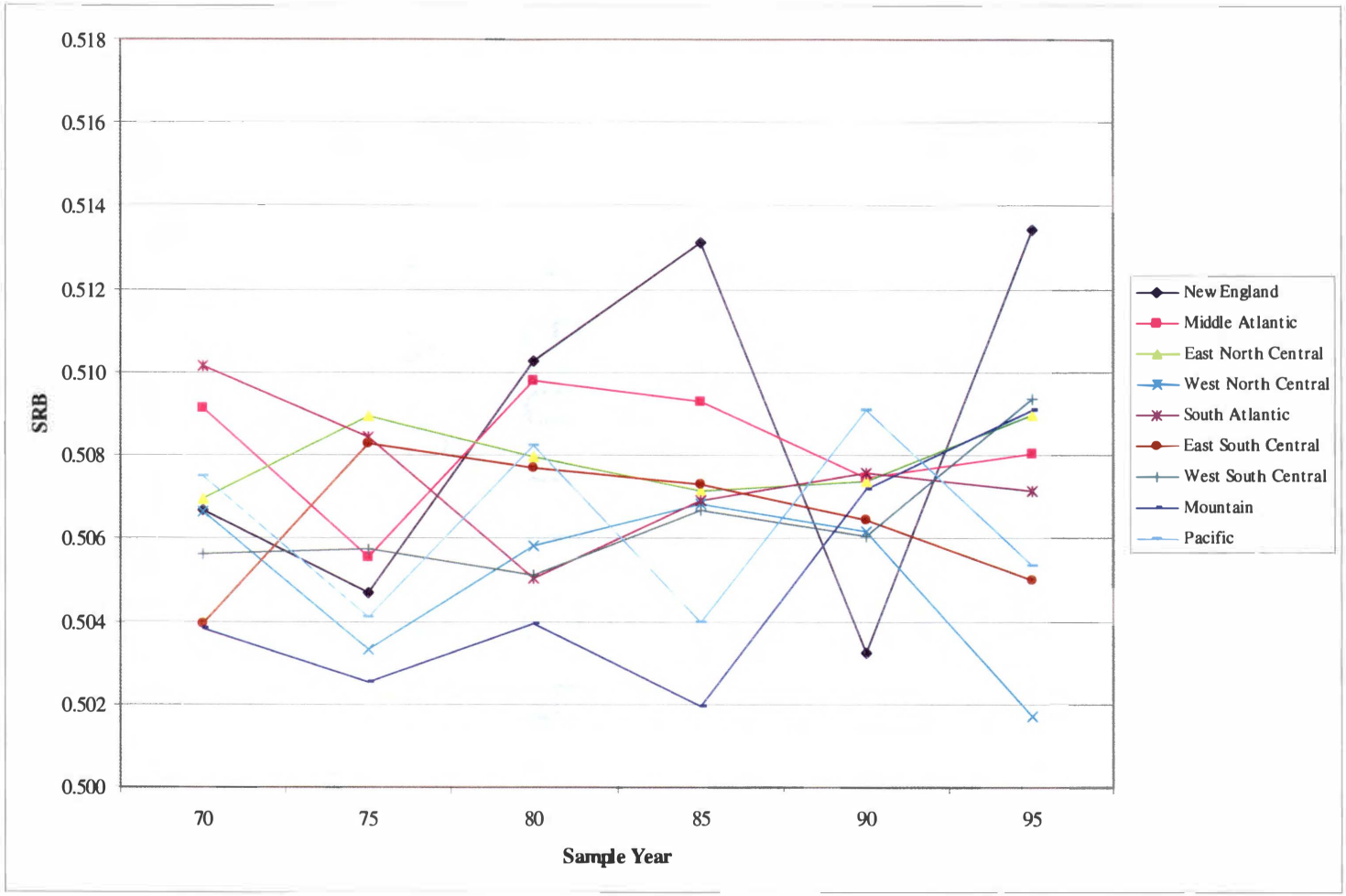
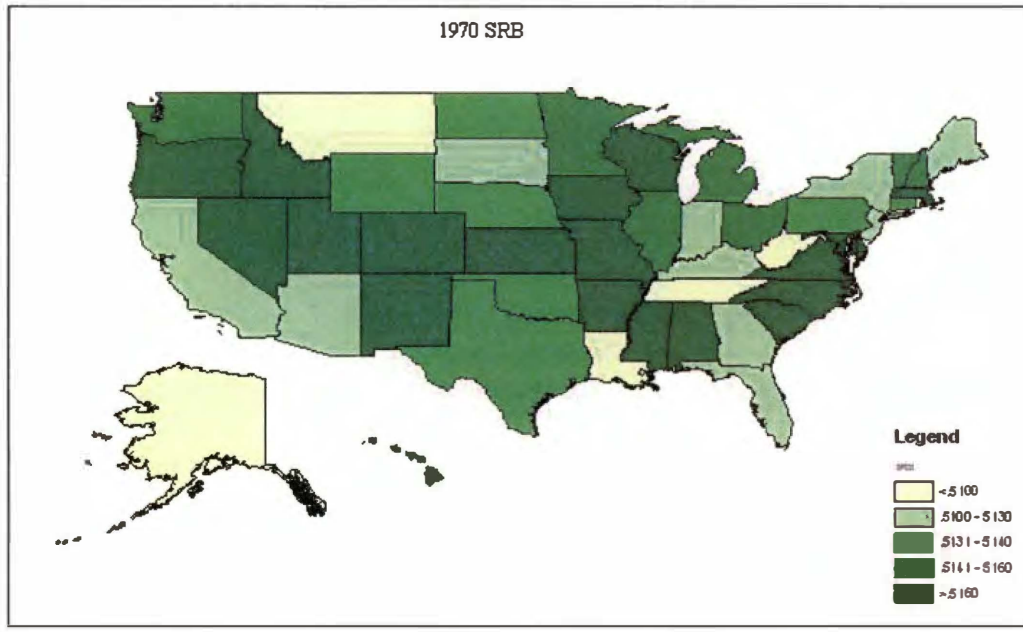


Figure 6. Black SRB (Male Proportion) by sample year and division in study period dataset



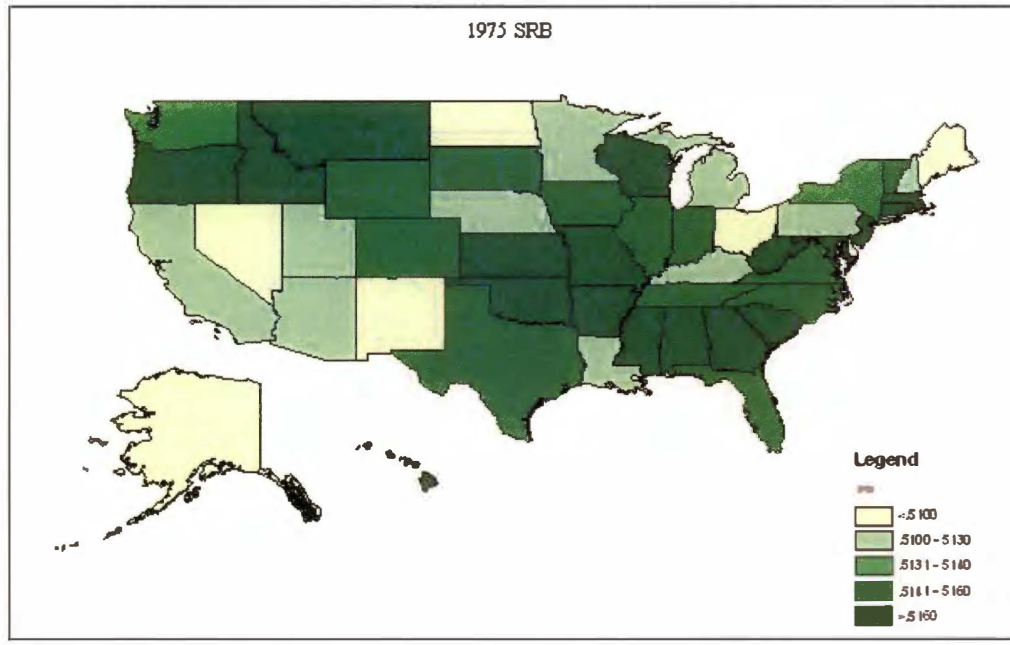
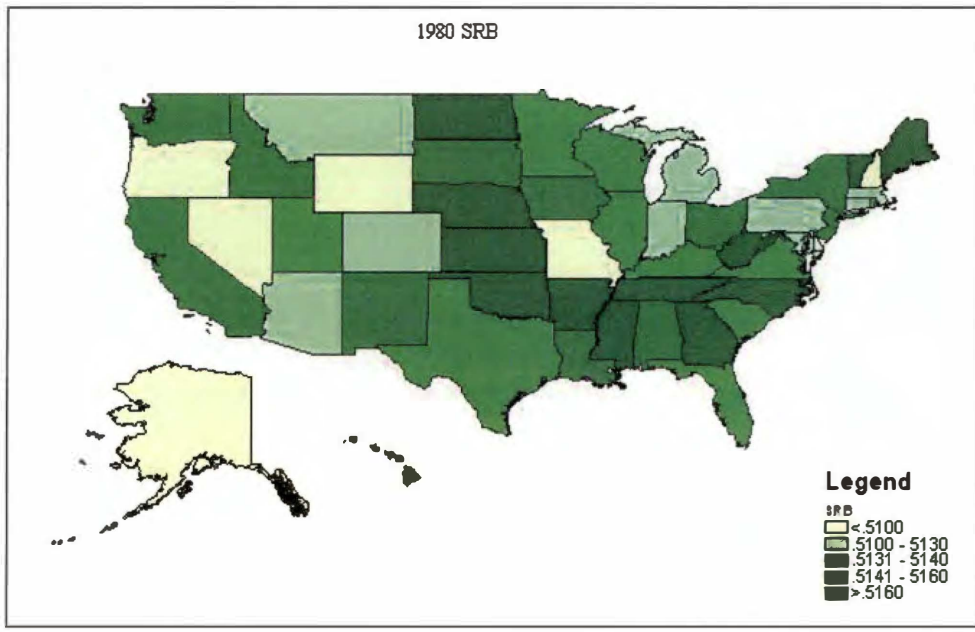


Figure 7. White SRB by state 1970–1995 (a) 1970-1975



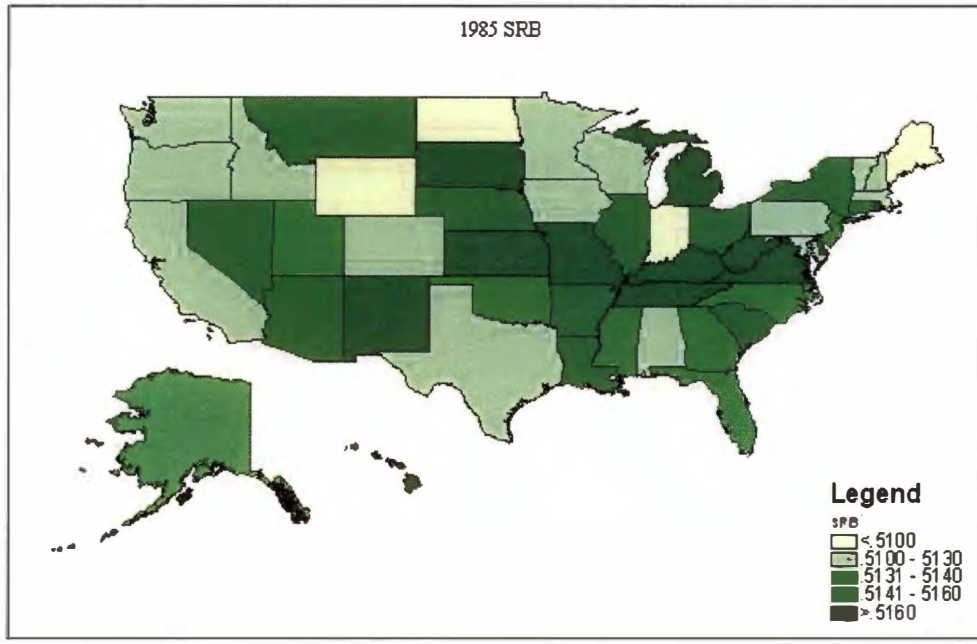
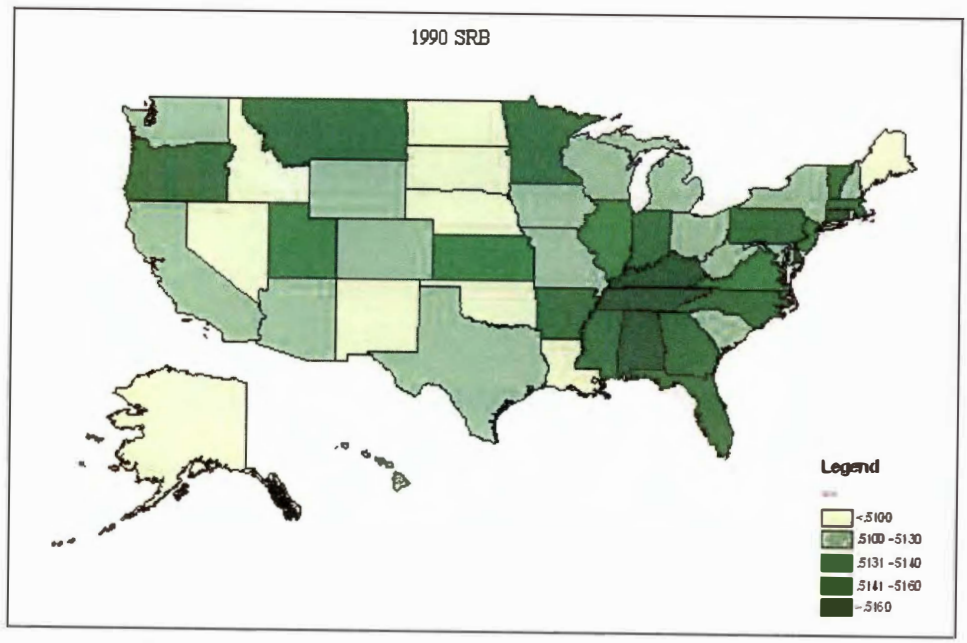


Figure 7. Continued (b) 1980-1985



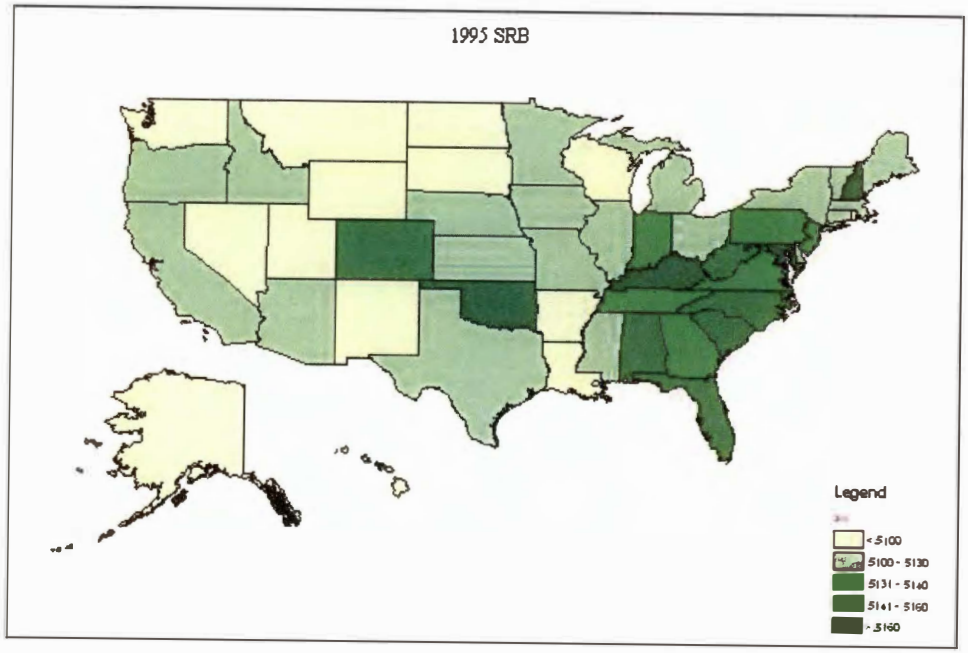
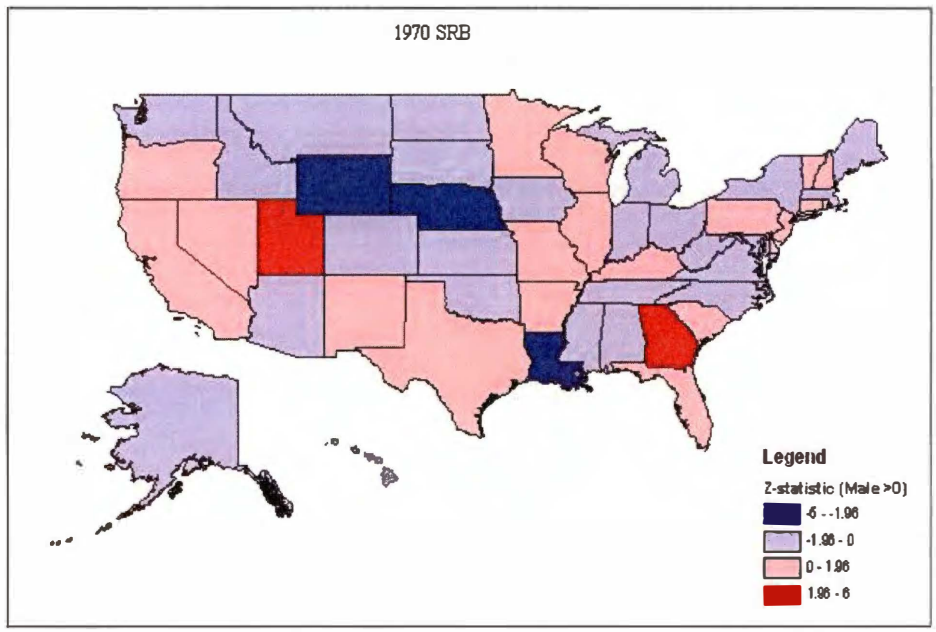


Figure 7. Continued (c) 1990-1995

Table 4. Significant Z-statistic White SRB Values for States

State	70	75	80	85	90	95	All Years
Tennessee	–1.547	0.657	3.080	1.596	1.917	0.558	2.857
Alabama	2.591	1.119	0.780	–0.509	1.919	1.197	2.776
Georgia	–1.273	2.459	2.329	0.099	1.124	0.419	2.206
South Carolina	1.357	2.260	–0.048	0.771	–0.031	1.216	2.145
Mississippi	0.347	2.913	1.097	–0.034	0.770	–0.142	2.116
Virginia	0.799	0.394	–0.095	2.904	0.187	0.574	1.926
North Carolina	0.902	0.008	1.174	–0.229	1.036	1.790	1.916
Oklahoma	–0.435	1.469	2.611	–0.012	–1.219	1.840	1.909
Washington	0.046	–0.124	0.228	–1.295	–0.922	–2.620	–2.116
Louisiana	–3.143	–1.468	0.418	0.425	–1.665	–1.162	–2.270
Alaska	–2.706	–1.215	–1.785	–0.065	–0.602	–0.531	–2.516
Texas	–0.572	0.575	–0.787	–2.246	–2.010	–0.617	–2.574
New Mexico	0.959	–3.485	0.131	1.050	–2.137	–2.583	–2.613
Nevada	0.582	–1.030	–1.369	0.108	–1.468	–2.300	–2.723
California	–1.623	–2.807	0.789	–1.866	–0.344	–0.827	–2.744



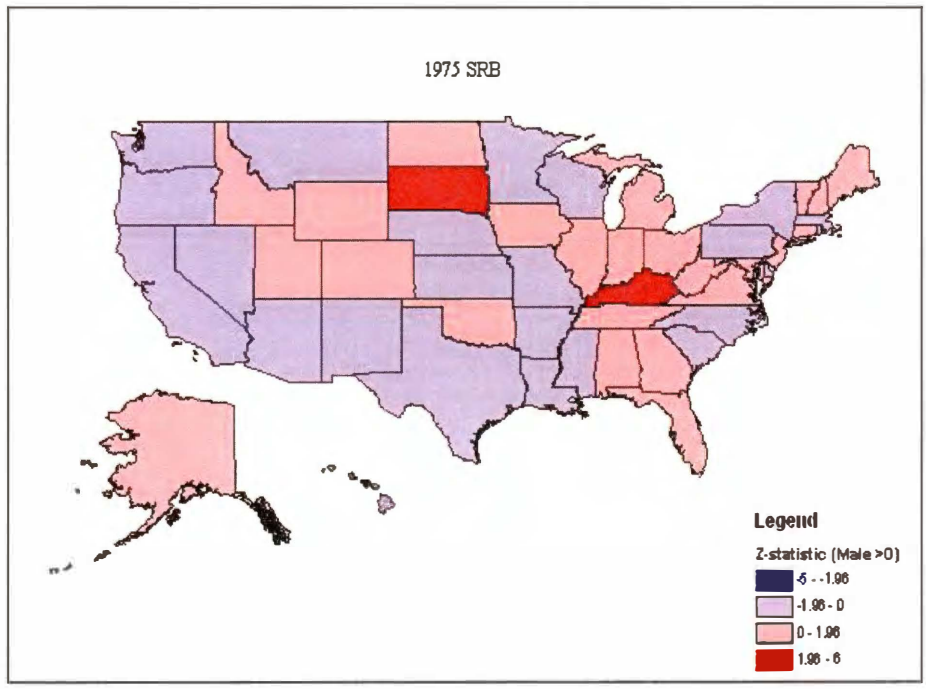
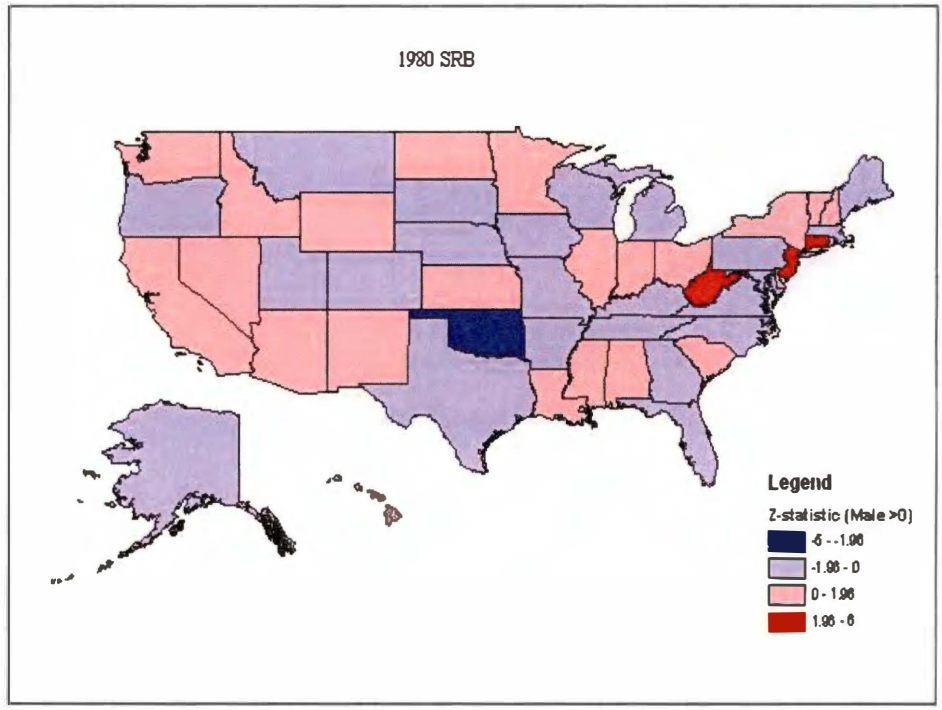


Figure 8. Black SRB by state 1970–1995 (Z- statistic)
(a) 1970–1975



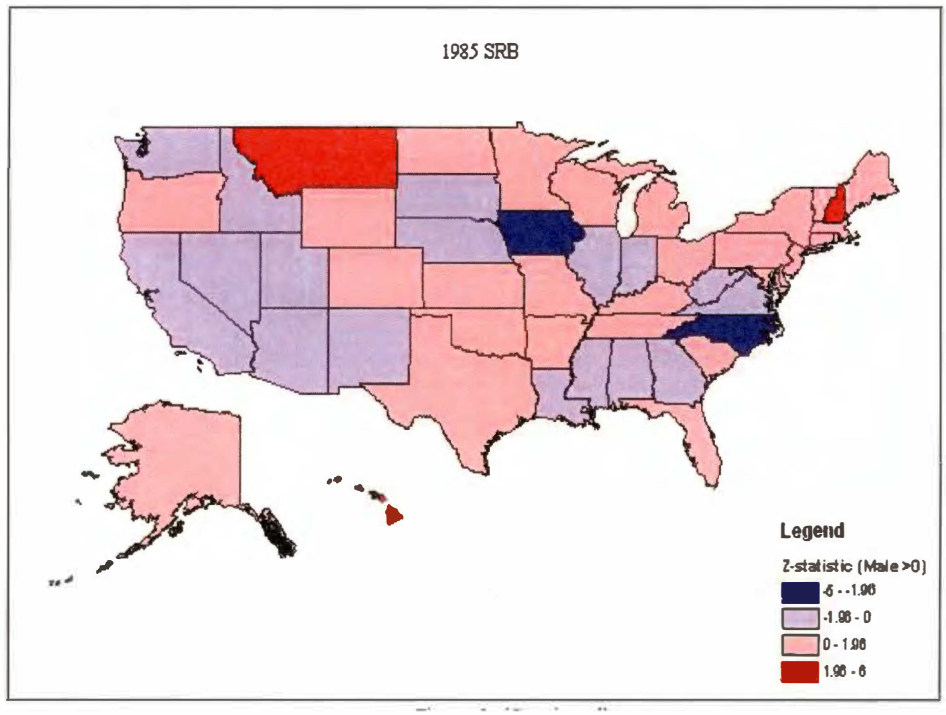
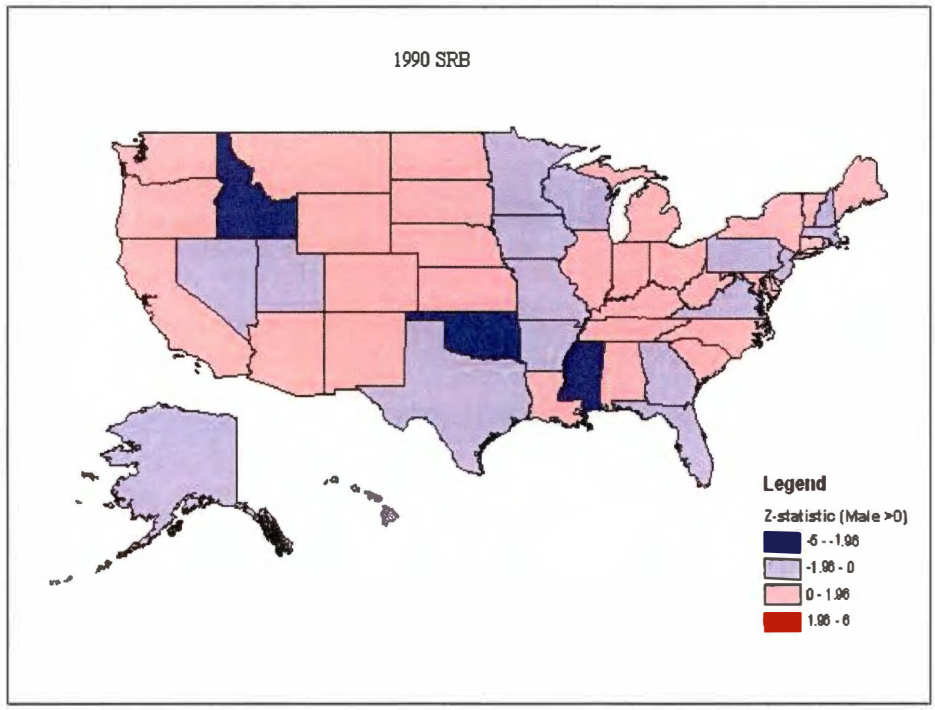


Figure 8. Continued (b) 1980–1985



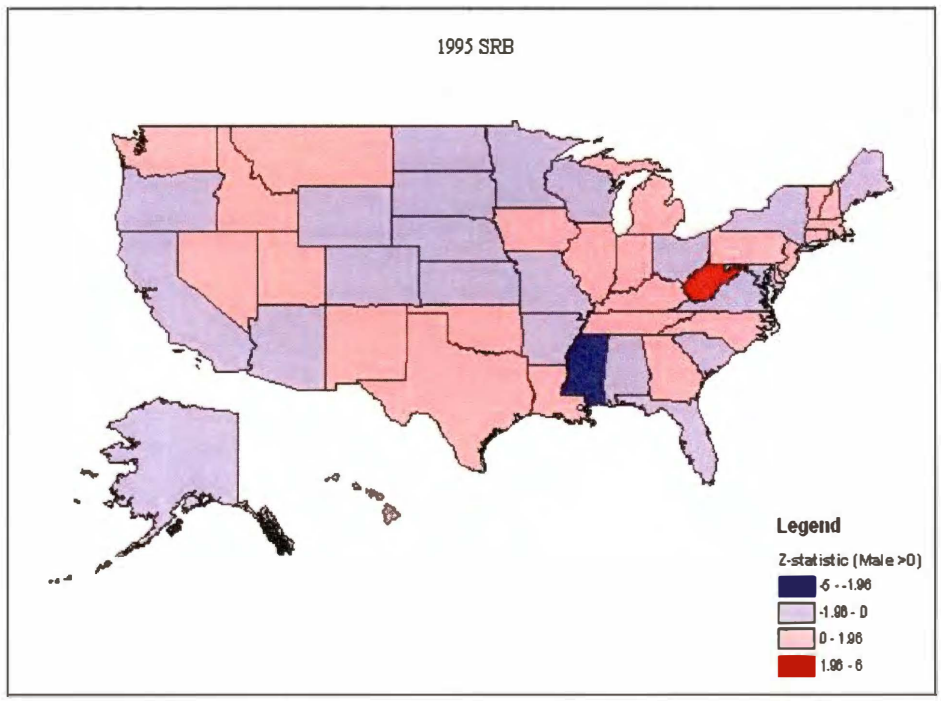


Figure 8. Continued (c) 1990–1995

Table 5. States with Significant Z-statistic Black SRB Values for Combined Birth Total.

	70	75	80	85	90	95	All Years
West Virginia	–0.494	1.896	2.499	–1.201	1.859	2.001	2.862
Kentucky	0.221	2.932	–0.771	1.177	1.552	0.143	2.146
New Jersey	0.900	0.243	2.181	1.454	–1.294	1.719	2.058
Mississippi	–1.475	–1.814	0.666	–0.777	–2.541	–1.976	–3.159

state with significantly high or low total black SRB is present on the list for white SRB Z-statistic values except Mississippi, which is, interestingly, significantly low for black SRB and significantly high for white SRB. Mississippi had the highest black population percent in the U.S. during the study period (approximately 35 percent).

Geographic distribution by U.S. county

There are currently 3,141 county and county equivalents in the U.S. (3,142 for those who count Kalawao County, Hawaii as separate from Maui County). To map SRB at the county level, I created a map of 3106 county areas that remain constant for all sample years. In some cases this required that contiguous county areas be aggregated and county birth data totaled for the joined area. I omitted Alaska because 1970 Alaska borough boundaries have subsequently been completely redrawn and share no common boundaries with current county areas. In anticipation of spatial autocorrelation analysis, I also omitted the four Hawaii counties from the mapping area, leaving only the 48 contiguous states. Details of this method are contained in the appendix.

To map spatial variation, I calculated Z-statistics for county-level white birth SRB by sample year (Figure 9). I also totaled all white births by county in the study period dataset and converted them to a Z-statistic for tabular analysis. Table 6 displays counties with more than 1000 combined total white births and having a significantly low SRB, as measured by Z-statistic. Six of the 14 counties in this category are in the Pacific or Mountain States and two are in Texas.

Table 7 lists counties with total births greater than 1000 and having a significantly high SRB for combined white births. These counties are mainly distributed in the east, with a somewhat higher representation in the southern U.S. than other regions. Only one county from west of the Mississippi, Honolulu, is among these counties.

I used the geostatistical analyst extension of ESRI ArcGIS 8.3 to prepare kriged quantile maps of the Z-statistic values for SRB by year. For all sample years, the ordinary kriging 0.5 quantile maps method is selected. I made no transformations and used the spherical model with neighborhood selection of at least five neighbors to construct a filled contour map for each of the six sample years. To allow better comparison, each kriged map uses an identical contour interval, displayed in the legend

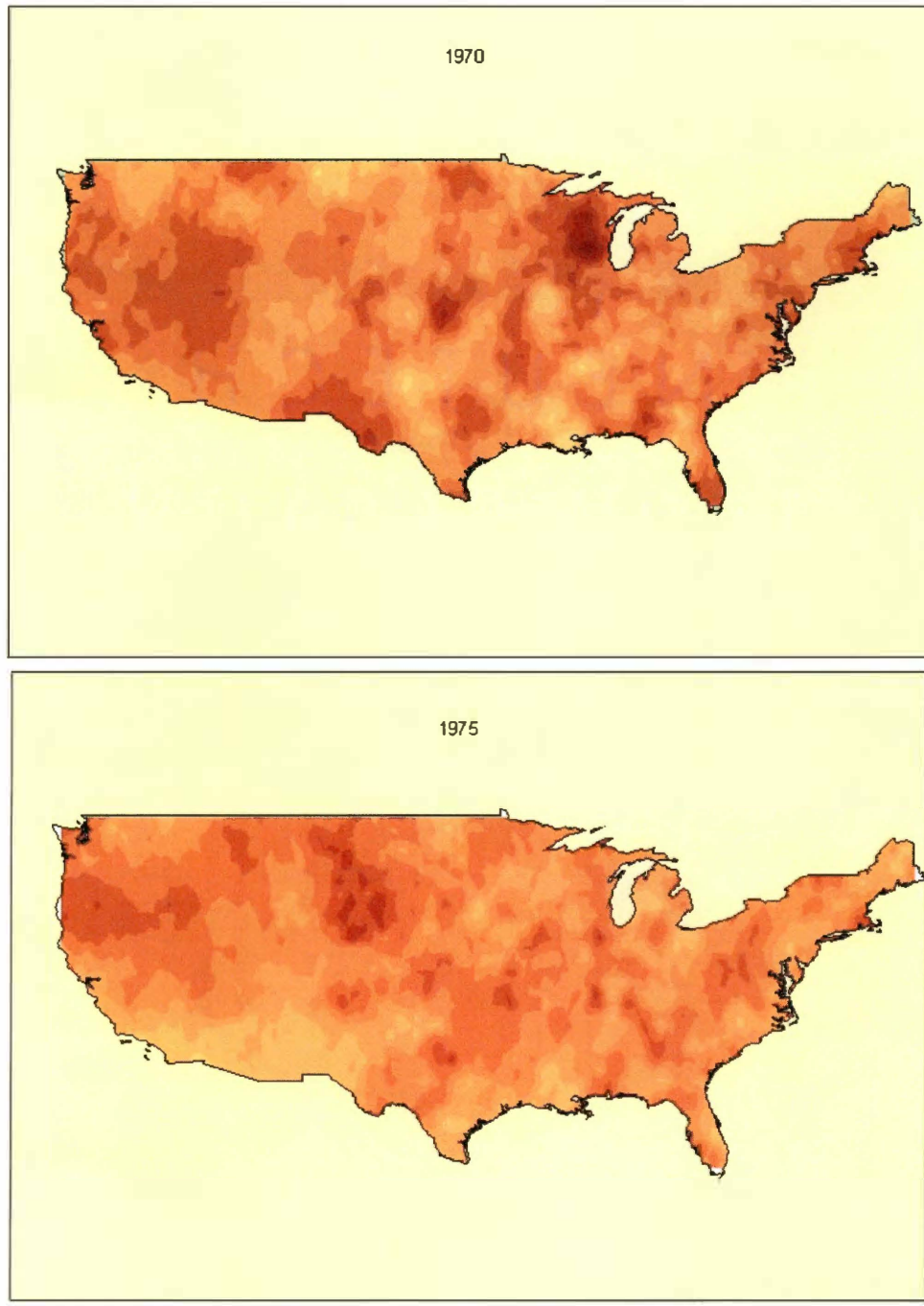


Figure 9. County SRB by study year (krieged Z-statistic surface)
(a) 1970–1975

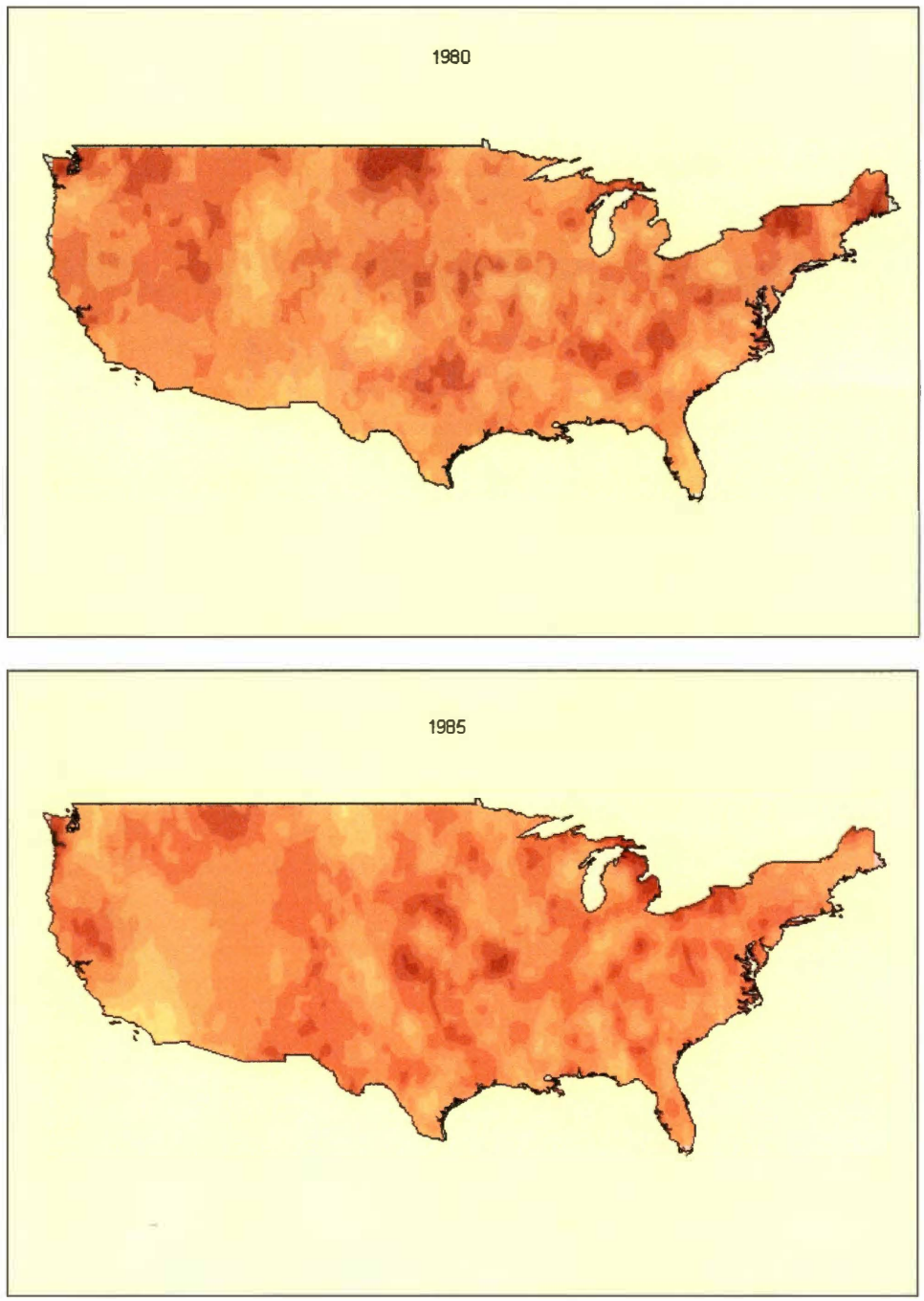


Figure 9. Continued (b) 1980–1985

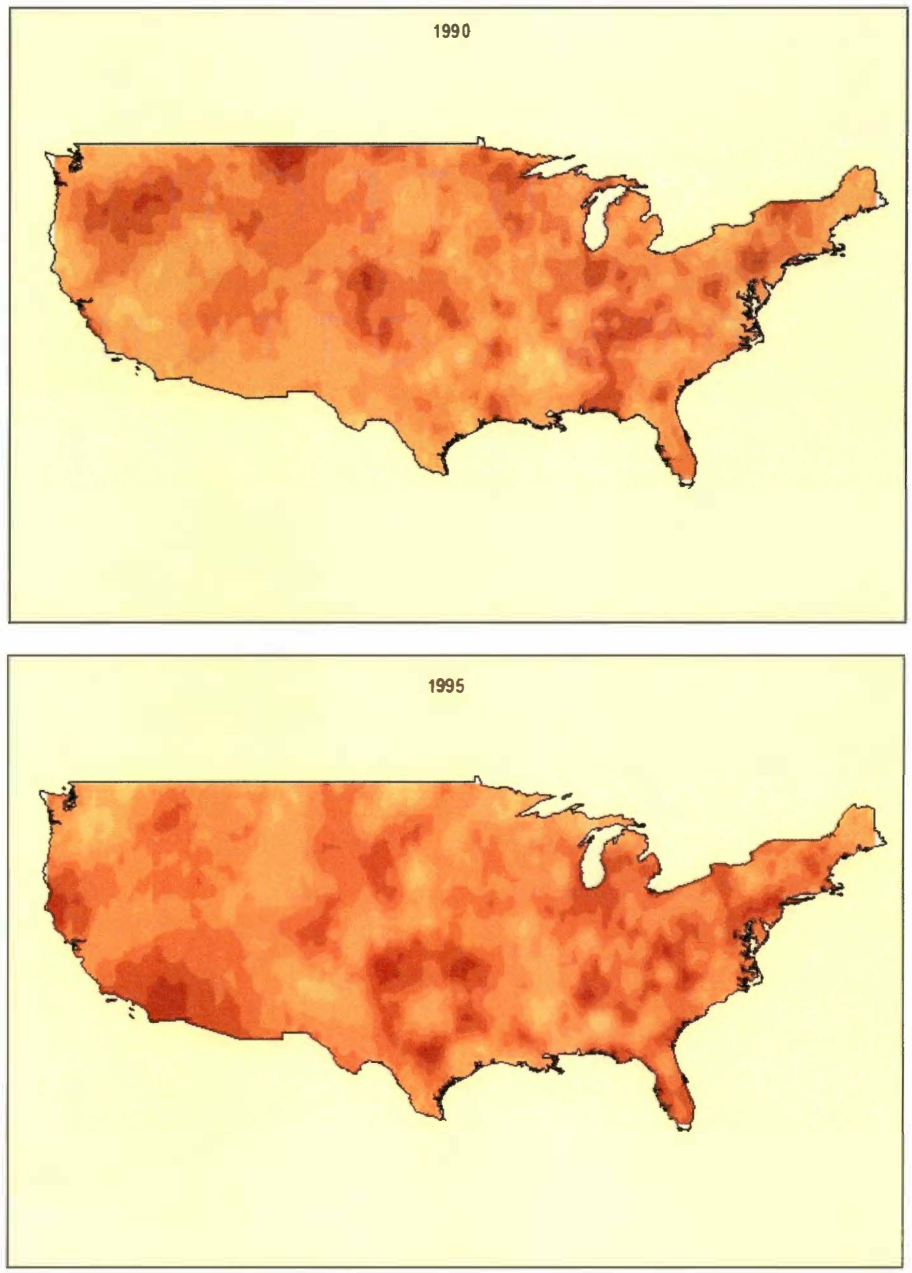


Figure 9. Continued (c) 1990–1995

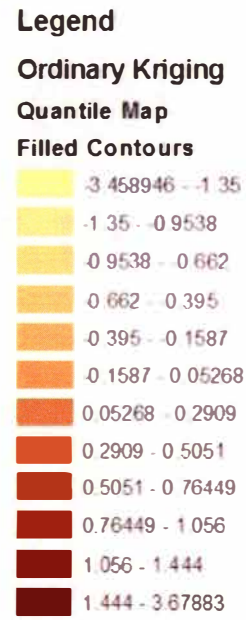


Figure 9. Continued (d) Legend

Table 6. Counties with Significantly Low White Birth Z-statistic and Combined Births Greater than 1000

County	State	Division	Total Births	SRB	Z-stat
King	Washington	Pacific	88829	0.5081	–3.2706
Churchill	Nevada	Mountain	14118	0.5012	–2.9351
Hidalgo	Texas	West South Central	48262	0.5069	–2.9233
Tulare	California	Pacific	29527	0.5055	–2.755
Bexar	Texas	West South Central	103938	0.5093	–2.7271
Los Angeles	California	Pacific	677639	0.5119	–2.7246
Ocean	New Jersey	Middle Atlantic	26445	0.5061	–2.4342
Anoka	Minnesota	West North Central	18526	0.5047	–2.4113
Yakima	Washington	Pacific	17446	0.5046	–2.3692
Atlantic	New Jersey	Middle Atlantic	12864	0.5034	–2.2988
Stanislaus	California	Pacific	29296	0.5069	–2.2792
Greene	Ohio	East North Central	9198	0.5027	–2.0786
Palm Beach	Florida	South Atlantic	36703	0.5083	–2.0245
York	Maine	New England	11563	0.5044	–1.9757

Table 7. Counties with Significantly High White Birth Z-statistic and Combined Births Greater than 1000.

County	State	Division	Total births	SRB	Z-stat
Harrison	Mississippi	East South Central	22006	0.5276	3.0733
Jefferson	Kentucky	East South Central	44941	0.5206	2.9777
Mecklenburg	North Carolina	South Atlantic	25519	0.5224	2.8267
Richmond	Virginia	South Atlantic	6712	0.5301	2.7119
Cumberland	New Jersey	Middle Atlantic	8865	0.5278	2.6854
Charleston	South Carolina	South Atlantic	16151	0.5239	2.6392
Tulsa	Oklahoma	West South Central	36267	0.5203	2.585
Kenosha	Wisconsin	East North Central	9943	0.5264	2.5635
Clayton	Georgia	South Atlantic	11861	0.5245	2.3841
Chester	Pennsylvania	Middle Atlantic	24209	0.5211	2.3461
Montgomery	Maryland	South Atlantic	41492	0.5192	2.3156
Honolulu	Hawaii	Pacific	24732	0.5209	2.301
Morris	New Jersey	Middle Atlantic	27986	0.5204	2.2935
Hudson	New Jersey	Middle Atlantic	35336	0.5196	2.2901
Jefferson	Alabama	East South Central	31617	0.5199	2.2511
Richmond	Georgia	South Atlantic	9312	0.5247	2.1524
Outagamie	Wisconsin	East North Central	11691	0.5235	2.1479
Madison	Indiana	East North Central	9697	0.5241	2.0744
Linn	Iowa	West North Central	14219	0.522	2.0272
Dutchess	New York	Middle Atlantic	16238	0.5214	2.0091

following the six maps. These intervals were based on an automated evaluation of natural groupings of Z-statistic values in the 1995 dataset that assigns breakpoints where there are relatively large jumps between these groupings (the "smart quantile estimation" method of ArcGIS 8.3). Because the Z-statistic uses the population SRB mean for the year of sampling, each of these maps displays for the year of study the relative concentration of higher than average births of girls (lighter colors) and higher than average births of boys (darker colors).

Figure 9 shows no consistent geographic pattern of SRB concentrations over the study period at the geographic scale of the U.S. county. SRB contours are skewed by the larger sizes of counties in the west, but, even accounting for this, one is hard put to infer a stationary front of SRB values. Some consistency in the presence of low-SRB patterns in areas of Washington and Louisiana can be seen in a majority of years, for example, but more apparent are polar changes of SRB values in the same region from year to year. In the Southwest, for example, SRB values are significantly high in 1970 and significantly low in 1975. The safest observation is, as Gini (1955) observed, that the SRB varies by time and place. Although the local geographic variation shown in Figure 9 controls for racial distinctions by considering only white SRB, there are apparently factors besides race that cause SRB to vary over time in a particular place.

Statement of the problem

My survey of the geographic character of the U.S. SRB reveals some regional differences that appear to have persisted through the study period. Declines in the SRB appear to have occurred in most U.S. regions, although less so in the southern U.S. and more so in the western U.S. than elsewhere. These regions both share the distinction of having higher population growth during the 1980s than did other regions (U.S. Census 2002). County-level mapping reveals that SRB values vary markedly in specific locations from year to year, with no consistent environmental or demographic pattern evident. The decline in total U.S. SRB from 1970 to 1995 is mirrored by a decline in the mean SRB of U.S. counties, with the county mean SRB even more pronounced than the total U.S. SRB. (Figure 10). To answer why there is a greater decline in mean county

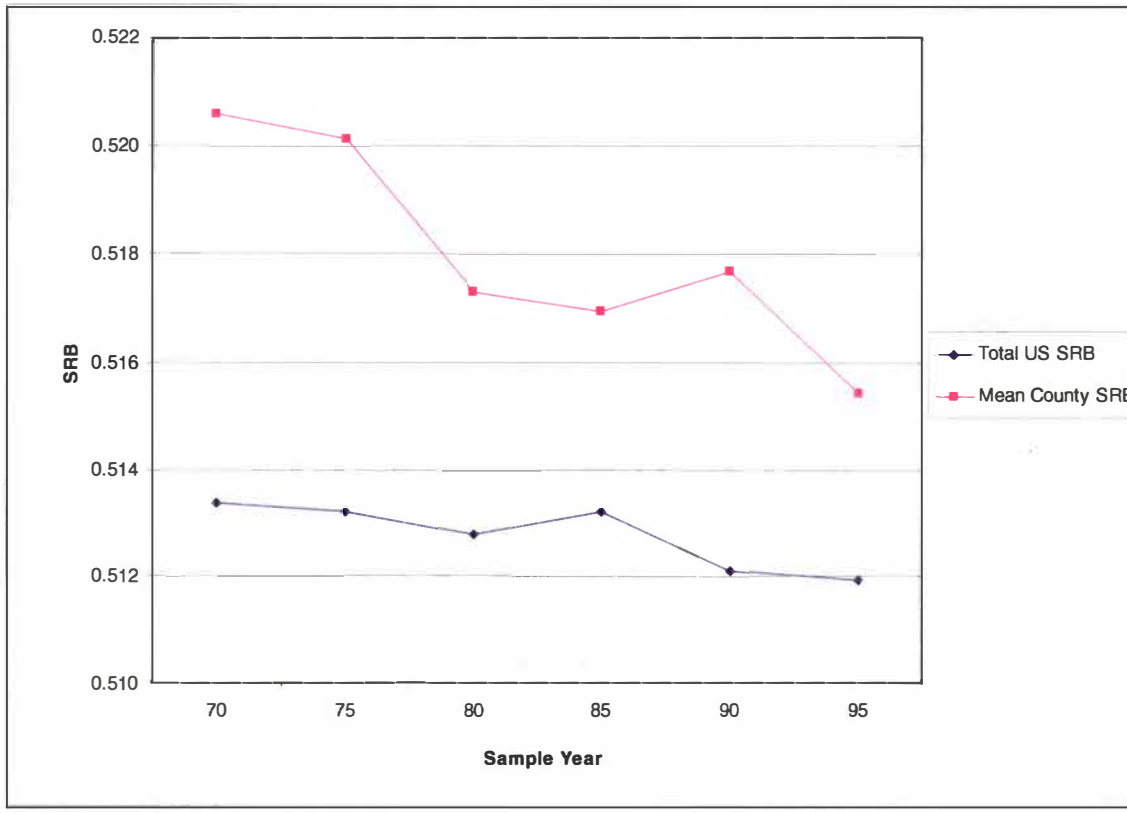


Figure 10. Mean county SRB by sample year vs. total U.S. SRB

SRB compared to the U.S. average requires an evaluation of SRB influences at local geographic scales.

Undermining a hypothesis of environmental toxin influence are the differing geographic patterns of SRB trends among white and black racial groups. Why these patterns vary in space and time is the central problem of SRB research. Further geographic analysis of SRB is not likely to provide much enlightenment about the cause of this spatial and temporal variation without consideration of the social and biological characteristics that might cause SRB to vary within groups and among individuals. Lazarus (2002) attributed our failure to decipher the SRB in past studies to three factors: The first is a methodological failure to control for confounding influences. Allen et al. (1997) did not adequately address, for example, the difference in racial SRBs in their regional analysis of decline in the U.S. and Canada. The second is the lack of an adequate conceptual theory of the biological mechanisms of human sex determination that might help in understanding results. Finally, there is little understanding of mechanisms that effect the mediating of different processes that influence the SRB, compounded by the fact that biases away from normal tend to very small.

The problem addressed by this thesis is therefore to develop and apply a geographic analysis method that adequately considers demographic and environmental influences on the SRB. Before attempting more rigorous analysis that addresses methodological pitfalls, I review in the following chapter previous research into sociodemographic and biological factors that may affect the SRB.

Chapter 2. Literature Review: The Nature of the Human Sex Ratio at Birth

Suboptimal prenatal conditions and the vulnerable male

Ronald Chambliss, whose geographic analysis of the U.S. SRB in 1949 has already been described, surveyed in the same study varying opinions from geographers regarding the impact of climate, humidity, temperature, altitude, food, precipitation, and urban vs. rural residence on the SRB. He also described the possible influences on the SRB known to mid-20th century researchers interested in the topic: relative age of parents, fertility rate, religion, standard of living, frequency of coition of parents, and abstention by parents from sexual relations at certain stages of the menstrual cycle. Few of these potential causes have been rejected completely by researchers in the intervening half century of research, and many have been added.

Chambliss's geographic study used the same assumptions and methodology that informs most SRB research, which aims to seek and explain the causes of biases away from an apparent biological norm of equilibrium between males and females. As noted in Chapter 1, differences in SRB among races were one of the earliest such biases noted and remains the cause of the largest noted natural variation in the SRB (James 1994, Ruder 1985). As social research matured in the 1920s and 1930s, researchers began to attribute the lower SRB of blacks to their lower socioeconomic status, and to the general principle that conditions that weigh negatively on healthy gestation will lower the SRB (Winston 1931). This principle was based on the idea that the sex ratio at conception (SRC), also termed the primary sex ratio, is more male biased than the SRB. According to this hypothesis, poor nutrition, reduced or missing medical attention, and other consequences of lower economic status will cause a greater rate of stillbirths and spontaneous abortions that will proportionally affect male embryos more than female. Winston (1931) found evidence of higher stillborn rates among U.S. blacks than among whites; he also attributed the birth order effect to the lower socioeconomic resources of large vs. small families. Winston's 1931 hypothesis that suboptimal conditions in the womb lead to lower SRBs continues to be an organizing theory in SRB research,

accommodating both acceptance of the chromosomal theory of sex determination (Henking 1891) and the supposition that the SRC is higher than the SRB.

The SRC is not known in any population, but researchers generally still agree that it is higher than the SRB. SRC values of .545 [120] or higher for the sex ratio at birth are typically estimated based on stillbirth and abortion counts (Winston 1931; Carey and Lopreato 1995). Also, the sex ratio of preterm infants is higher (~.520 [110]) than that of full term infants (Cooperstock and Campbell 1996; Cooperstock et al. 1998). In chromosomally normal spontaneous abortions the male death rate is about 30 percent higher than that of females; also, females appear to develop sooner in the womb and survive preterm birth better (Ingemarsson 2003). Generally, males die earlier and more easily than females in the womb (Naeye at al. 1971, Ingemarsson 2003), and many SRB researchers have assumed that any factor that reduces the quality of the prenatal environment would lower the SRB. This principle applies to psychological stress as well as physical factors, and could be used to explain the lower SRB among infants whose mothers smoke (Fukuda et al. 2002), or who are exposed to heavy smog events or floods (Lyster 1974), or earthquakes (Fukuda et al. 1998), or who have experienced recent deaths of close family members (Hansen et al. 1999), or live in active combat zones (Zorn et al. 2002).

However, this explanation falls short of being a unifying theory of the causes in the variations in the SRB. It does not explain, for example, why the SRB rises with increased frequency of coitus between parents (James 1997b, Martin 1997), or during the period during and after wars in nations involved in combat (Graffelman and Hoekstra 2000), or among children of fathers who have prostate cancer (Bosland 1988). In this chapter I will summarize such factors and the postulated underlying mechanisms presented in the prodigious literature on the SRB. The factors I will consider range from very small fluctuations in large populations to very large differences in small populations of parents with unique characteristics or experiences.

Family factors

In addition to racial differences, dozens of articles are devoted to describing the relationship of SRB to demographic factors readily available on hundreds of millions of

birth certificates: maternal age, paternal age, month or season of birth, plurality (single or multiple births), and birth order (i.e., the numeric order of the mother's pregnancy, sometimes expressed as maternal parity—the number of previous births by the mother). These and related characteristics are variously termed as intra-population, birth certificate, sociodemographic, individual, or "family" factors, and all are assessed as influencing SRB at a lower magnitude than racial factors. I will adopt the term "family factors" to distinguish these specific individual birth factors from somewhat broader demographic characteristics examined later in this review. Aged mothers (Juntunen et al. 1997; Orvos et al. 2001), aged fathers (Jacobsen et al. 1999, Nicolich et al. 2000) and later birth order (Erickson 1976, and others) have all been identified as conditions leading to lower SRBs and as support for the theory that less optimal conditions reduce the SRB from its norm. One of the most intractable problems with analysis of the effects of paternal age, maternal age, and birth order is that they are highly correlated, and various studies have examined them both univariately and multivariately.

There appears to be strong support for if not consensus about the existence of an SRB birth order effect. The later in the birth order a child is born, the less likely that it will be a boy. Jacobson et al. (1999) did not find a significant univariate effect of birth order (or maternal age) on the SRB of 800,000 Danish births during the period 1989–1993, while Erickson (1976) found that birth order was significant—but the age of either parent was not, once birth order was controlled for. However, most authors have found that birth order significantly influences SRB either as a univariate factor or in combination with other factors. Lazarus (2002) found that birth order significantly lowered the SRB in 81 percent of 16 reviewed studies, paternal age in 62 percent, and maternal age in six percent.

Declining SRB has been found in association with increasing maternal age in combination with birth order (Juntenen et al. 1997, Orvos et al. 2001), and a maternal age affect has also been found independently of birth order (Ruder 1985). The relationship between maternal age and SRB may not be linear. Ulizza and Zonta (1995) found that a quadratic function of the proportion of males among first-born children and mother's age was "a fairly good predictor" of the SRB in the wider population. James and Rostron

(1985) found a curvilinear relationship between SRB and maternal age in England and Wales 1968–1977; Tarver and Lee (1968) also found that the relationship between maternal age and SRB was curvilinear in U.S. births, increasing sharply with age. The maternal age effect may therefore be more difficult to detect than paternal age and birth order because of its nonlinear relationship with SRB.

More studies appear to favor paternal age over maternal age in the magnitude of its influence on the SRB; James and Rostron (1985) found that linear declines are associated with increasing birth order and increasing paternal age, and this relationship has been found in several studies. Ruder (1985) found that increasing paternal age significantly reduced SRB in U.S. births independent of birth order, and had a more significant effect than maternal age. Jacobson et al. (1999) found that the SRB of Danish births (1989–1993) born to fathers aged 24 and younger was .516 [106.6], but dropped to .510 [104.1] for fathers 40 or older. There is also some suggestion that family-factor effects differ among races. Nicolich et al. (2000) found that paternal age was a strong influence on SRB in U.S. populations, and stronger in nonwhites than whites; paternal age was twice as strong as maternal age in nonwhite births, but equal to maternal age in white groups.

Studies have also found that the age difference between mother and father was also a significant factor in SRB. Manning et al. (1997) found that fathers with mates five years or more younger then themselves have twice as many firstborn sons as firstborn daughters, while women who had children with men five years or younger than themselves experience an opposite effect: twice as many girls as boys among firstborn children. They also found that the mean age difference between spouses increased during and immediately after the two World Wars, with older fathers/younger wives possibly contributing to the oft-noted increase in SRB during and after wartime. Astolfi and Zonta (1999b) found a similar increase in the SRB in a subsample of parents with a wide age gap (greater than 15 years). If there is a paternal age effect decreasing SRB, it may be mitigated by mating with younger mothers, but older mothers/younger fathers have more girls than would be expected.

In a review of SRB literature in 1988, Chahnazarian offered a summary assessment, of this "less than clear" relationship among family factors, that appears to still hold up in the majority of subsequent studies. Younger parents sire more boys, and there are more boys in lower birth orders. When both paternal age and birth order are controlled for in multivariate studies, maternal age weakens or is not significant as an influence. The maternal effect also appears to decrease with increase in sample size. The effect of all these variables on SRB is small, and the stronger racial effect persists independently of other variables. To explain the reason for family factor effects, authors often note that increases in stillbirths are associated with increases in maternal age and birth order, and thus disproportionately increase the male fraction of prenatal mortality.

The importance of studies of parental age and birth order factors to the causes of SRB variation is discounted by James (1987a), who notes that almost all of the variation in SRB from these effects falls between the values of .516 and .513. He states that "...apparently these variables are not 'close' to the causes of variation of sex ratio, and so are unlikely to yield any useful clues to those causes." He cites examples of much greater variation in SRB among parents in special circumstances that may yield better clues about the underlying mechanisms of SRB. These and other findings regarding significant variations of SRB are reviewed here.

Plurality

Multiple birth—also termed "plurality"—has been found to have a significant influence on SRB. The SRB of twins (both monozygotic—identical, and dizygotic—fraternal) is lower than that of singletons (James 1987a, Jacobsen et al. 1999b). The SRB for triplets is also lower than that of singletons (Jacobson et al. 1999b). NCHS (1992) also found that the U.S. SRB of multiple births in the mid-1980s was lower (.501 [100.1]) than that of singleton births (.512 [105.1]); this difference was stronger among white births than black births. The relation of twinning rate to SRB has interested some investigators, particularly those seeking to explain racial differences in SRB. In general, the twinning rates of black populations in the U.S. are much higher than the twinning rates of white U.S. populations, although the gap has narrowed in recent years (NCHS 2002). Different twinning rates between white and black populations may also be present

in other nations. Nylander (1981) found significant differences between the twinning rates of a white population in Aberdeen, Scotland compared to the much higher rate of a black population in western Nigeria (12 incidences of twins per 1,000 births vs. 50 per 1,000, respectively).

The lower birthweight and greater incidence of preterm births among multiple births (CDC 1997) suggests another category of suboptimal prenatal conditions causing disproportionately greater male mortality. However, seasonal variation observed in the number of dizygotic births and in SRB values (James 1987), as well as the difference in twinning rates between black and white populations, introduces issues that are not simply explained by the principle of disproportionate prenatal male mortality.

Season of birth

Another family factor, season of birth, is readily available on birth certificates. Apart from SRB, the total *number* of births by season has long interested demographers, who have identified remarkably consistent patterns among populations. These patterns vary geographically and through time. A common pattern of either a spring peak (as in Europe and Canada) or a spring trough (as in the U.S.) and a September peak in either pattern has been found to occur regularly (Doblhammer et al. 2000). These patterns have been related to photoperiod/latitude (James 1990a, Roenneberg and Aschoff 1990) or local weather patterns (Lam and Miron 1996), suggesting that humans may have an innate biological tendency to adjust births seasonally to coincide with optimum nutrient availability and temperature. Brewis et al. (1996) found examples of nonseasonality in births in equatorial regions, consistent with the Bailey (1992) theory that seasonal birth changes are triggered by wide variations in temperature and food availability during the year. There is evidence that the biological triggers from seasonality, if they exist, may have been blunted by more homogeneous climate conditions in recent years and by modern living and working patterns (Doblhammer et al. 2000, Seiver 1985). Seiver (1985), for example, found that the seasonality of births in the U.S. South had been changed by the introduction of air conditioning—as it became more pleasant to have sexual intercourse in the summer, the previously documented April trough in births became less pronounced.

While the pattern of higher conceptions in winter vs. summer has flattened out in the last 100 years (Doblhammer et al. 2000), there remains some element of SRB variation by season that also varies over time and by geographic location. Studying SRB over several decades, James (1984, 1987a, 1990a) found a seasonal pattern of births in European countries of a major SRB peak in the spring and a minor SRB peak in the fall. However, he discovered the opposite was true in the U.S., with a minor peak in the spring and a major peak in the fall, which could also be described as one broad peak from summer to early fall. He found, however, that the pattern of England and Wales in the last 20 years has changed to resemble the U.S. pattern and believes that such is also true in other European countries. It may be that SRB seasonality variation has evolved to be responsive to seasonal advantages, and may in some way still respond to climatic and weather cues. Successful physiological adaptations to some remote evolutionary past, such as the Pleistocene African savannah, may still be a part of the human DNA. Many activities of the endocrine system, particularly, have puzzling origins.

The human endocrine system has displayed a significant number of seasonal and circadian mechanisms related to certain of its functions (Hansen et al. 2001). To explain seasonal birth variability, Bailey et al. (1992) used a model linking rainfall to food production and then to fertility via an ovarian nutritional function controlled by the pineal gland. The primary function of the pineal gland appears to be the production of melatonin. Seasonal and daily fluctuations in melatonin production have been documented, relating to the inhibiting factors of light on the activity of pineal gland. In addition to regulating sleep patterns, melatonin is known to influence sexual maturation, skin pigment changes, and the menstrual cycle and other body rhythms. In women, melatonin is associated with seasonal variation observed in the concentrations of gonadotropins, hormones produced by the pituitary gland to regulate reproductive function, and of other gonadal steroids (Kiely et al. 1995, Wojtowicz and Jakiel 2002). James (1984, 1987b, 1990a) has found that the seasonality of SRB has been synchronous with the seasonality of dizygotic twinning and the births of boys with cryptorchidism (undescended testicles), both of which are conditions that appear to be positively associated with levels of gonadotropins in the maternal hormonal environment.

It is safe to make the generalization that human fetuses conceived in different seasons may be subject to very different intrauterine hormonal environments (Geschwind and Galaburda 1987). A number of animal studies have focused on the association of fluctuations in reproductive control hormones and seasonal breeding behaviors designed to deliver offspring in the most advantageous food availability and temperature conditions. Many of these studies show that the sex ratio of offspring varies with season and longer term variations in food availability and population stresses. This facultative manipulation of offspring sex ratio in response to changing environmental conditions is explained by Trivers and Willard (1973) as an adaptation by parents to increase their reproductive success. According to this and other sex allocation theories, which I will explain more fully later, mothers in good condition will more likely produce males and mothers in poor condition will likely produce females, especially in populations where males must compete strongly for mates. A corollary of this theory is that more females will be born in general in poor *nurturing* conditions such as periods of lower temperature or scarce food availability. Offspring born in poor nurturing conditions will likely be in poorer condition when they reach breeding age than those born when the weather is warmer or more food is available. Therefore mothers giving birth during these poor-condition periods will optimize their reproductive success by producing daughters.

While humans are not thought to be seasonal breeders, the evidence of seasonal variation in the total number of births suggests that they might have been when human survival was more directly tied to seasonally changing resource availability. How parental hormonal levels might influence the human season of birth is a mechanism not fully worked out. Cagnacci et al. (2003) did not observe the season of birth to be significant to SRB, but did find that the season of conception was, possibly because gestation period varies slightly and males may have different gestations compared to females: September to November conceptions favored boys while March to May conceptions favored girls. Lerchl (1999) found that SRB was partially correlated with environmental temperatures when he examined SRB in Germany from 1946–1995. Monthly temperature deviations from the overall mean for the 1946–1995 period were positively correlated with rises in the SRB when temperatures were time-lagged with the

SRB data by minus 10 or 11 months—suggesting that sex selection may have occurred *before* conception. The findings of both Lerchl (1999) and Cagnacci et al. (2003) support the idea that more males than females are conceived in seasons that portend more favorable environmental conditions during early nurturing of a newborn. Human SRB may therefore be partly influenced by internal rhythms mediated by anticipatory hormonal responses to environmental conditions. Cagnacci and Volpe (1996) and others have cited the seasonal variation in the parental internal hormonal environment as a line of evidence for this mode of control.

From variation in seasonal human SRB and other evidence, James (1986, 1987b) was the first to postulate that the SRB in humans was partially controlled by the parental hormonal environment at the time of conception. The prevalent theory that variations in SRB were accounted for by physiological constraints such as older wombs or aged sperm and consequent excessive elimination of the male lacked an evolutionary explanation of why the SRC was higher than the SRB. The hormonal environment theory may serve as the foundation of more complex and flexible explanations of variation in human SRB. With the rise in evolutionary biology and the remarkable success of sex allocation theory in explaining the SRB of some species, some authors have sought to apply the principles of sex allocation to human populations. While such theories do not generally discount the possibility that there are nonadaptive, excessive losses of prenatal males due to poor nutrition or other stressors, they suggest that some variations in the human SRB may be due to facultative, adaptive manipulation of SRB for reproductive advantage. Adaptive control implies a mechanism of hormonal response to environmental conditions such as local temperature deviations and is the underlying basis for the argument that environmental toxins similar to sexual hormones have disrupted the normal operation of this mechanism. Evaluating this theory requires a review of the biology of sex determination in humans and of the underpinnings of the sex allocation models, along with an examination of documented extraordinary deviations from SRB that might support such theory.

Sex ratio at conception vs. sex ratio at birth

Although the basic principles of genetic sex selection have been known since Mendel, the biological processes occurring at the time of conception are a slowly unraveling mystery. Mendel's first principle is that gametes of the parents are one half of a pair of alleles. On both the ovum and the sperm the sex-bearing chromosome is only one of twenty-three chromosomes (Henking 1891). On the ovum in humans, it is always an X-bearing chromosome. The sex-determining chromosome of the sperm may be either X or Y. The male zygote is thus always produced from the combination of a Y-bearing chromosome in the fertilizing sperm and an X-bearing chromosome in the ovum. Male heterogamety, the determination of a male in a species using two different sex chromosomes, prevails in all mammals; female heterogamety is the case in all birds. Fish, reptiles, and amphibians have mixtures of both these modes, and diverse mechanisms operate in invertebrates, with cases of varied sex determination mechanisms in a single species, or even changes in sex by an individual during a life cycle. In some fish and amphibian vertebrates and in many invertebrate species, sex may be determined *after* conception in response to differing photoperiod, temperature, pH (in aquatic species), nutrition availability, mate availability, and other social or environmental conditions. The genetic or chromosomal sex determination mechanism of humans and most vertebrates is thought to limit the parent's capability to manipulate the offspring sex ratio, but I will explore some challenges to this assumption.

The ratio of Y to X chromosomes in sperm would be expected to be a factor in the SRB, and might explain, among other influences, the paternal age effect. An excellent summary of sperm sex-chromosome bias studies is provided by Lazarus (2002). Some researchers suggest that the ratio of X to Y chromosomes in human sperm is about equal (Martin 1995), as would be expected by Mendelian segregation. Graffelmann et al. (1999) found no systematic sperm ratio bias in a small sample of European Caucasian men aged 23 to 56. Similarly, Irving et al. (1999) did not find that fathers who had three or more sons had significantly high numbers of Y chromosomes in their sperm, nor that those who fathered daughters had high numbers of X chromosomes. However, in another small sample, Bibbins et al. (1988) found that fathers that sired only girls had a

significantly higher proportion of X-bearing chromosomes in their sperm than a control group.

While there is only limited evidence that sperm sex-chromosome ratios might influence SRB, it appears true that sperm has been shown to vary in quality within the same individual. Fisch and Goluboff (1996) speculate that annual changes in temperature and climate may account for sperm count variations, with the highest sperm counts in the winter months. Abstinence before ejaculation may increase the proportion of X chromosomes (Hilsenrath et al. 1997), suggesting a mechanism for explaining why more frequent coitus might result in a higher number of males (James 1997b). Psychological stress may also affect sperm velocity and percentage of motile sperm, as in the case of men experiencing the death of a close family member (Fenster et al. 1997). This suggests that differences in sperm ratios could be mediated by hormonal responses to external factors.

Compared to the studies of sperm success in fertilization, the above-cited studies of sex-chromosome ratios in sperm and their effect on the SRB are few in number and more work using recently developed detection measures needs to be done before the question is resolved. The question is important—if there is facultative control of sex ratio, which parent controls it and how is it done? Perhaps the female has greater influence, given that sperm enter an environment under her control. Coital frequency, the timing of insemination relative to the menstrual cycle, and the age of the ovum are believed to influence the SRB, indicating that the intrauterine hormonal environment may be more hospitable to Y-chromosomes at certain times than others. Gray (1991) found that fewer males than females were conceived during the ovum's optimum fertile time and more males are conceived at either end of the ovum's fertile cycle, a U-shaped regression proposed in earlier studies by Guerrero (1974). Guerrero (1974) found in his reviews of animal studies that this regression held for "all known inseminations in animals."

Several factors affect whether an X- or Y-bearing sperm reaches the ovum, most notably the difference in the size and performance of the Y-chromosome-bearing sperm. Other than its task to determine sex, the function of the Y-chromosome is not well

understood. The human Y-chromosome contains only 78 genes, compared to almost 3000 in the X-chromosome. As such the Y-chromosome is smaller and lighter than the X-chromosome, so that the sperm carrying it has a more lively motility and is differently shaped, possibly allowing it to outrace the heavier X-chromosome sperm or better penetrate the cervical mucus when it is at its thickest. The cervical mucus thins considerably in the one or two days prior to ovulation, and then thickens immediately after. According to Martin (1997), the more ungainly X-chromosome sperm reach the ovum more easily during this time, while the Y-chromosome sperm have a relatively greater chance of success on other side of this period, the so-called U-shaped regression. Frequent coitus may also leave seminal fluid debris that reduces cervical mucus penetrability, further giving the Y-chromosome an advantage.

James (1997b) calculated the overall effect of coital frequency on SRB in a population as much less significant than Martin (1997) did. While Martin's hypothesis that increased coital frequency would produce more boys because of changes in cervical mucus penetrability, James (1997b) showed that increased coital frequency could be mathematically modeled to show more conceptions on the ends of the U-shaped regression, favoring boys. The supposition of coital frequency influences are also based on the "honeymoon effect" of high male conceptions from the Renkonen (1970) study of Australian births from 1908–1967. He showed that the SRB was .522 [109.4] for conceptions in the first month of marriage and .514 [106] in the following and subsequent months of the first year of marriage, which he attributed to the high coital rates of the first month (over 20), compared to subsequent months. Coital frequency also drops after the first year of marriage (or intimacy), although how much is debated. This paradigm has also been used to explain the birth order effect and paternal age effect.

According to James (1987a, 2003), frequency of coital activity may also account for the greater number of births observed during and immediately after wars in countries involved in the conflict, when coital activity is presumably higher during military leave (I will present some alternatives for the cause of the war effect later). The increase in SRB related to war has been suspected since at least the time of the Napoleonic wars, and

current evidence suggests that it is at least true in certain countries involved in conflicts, whatever its cause (Graffelman and Hoekstra 2000).

The differential success of the Y-chromosome during the fertilization cycle may also explain some aspect of the male vulnerability during gestation. James (1997b) believes that periods that favor male conception at the beginning and end of the menstrual cycle are suboptimal for beginning gestation, compared to the middle part of the cycle. This would partly explain why there is a higher SRB in spontaneously aborted fetuses, stillborn births, and neonatal death from suspected suboptimal intrauterine environments. The excessive prenatal vulnerability of males may be in some way inherent in the female's cyclical receptivity to the Y-chromosome.

The case for hormonal influences

The hormonal environment theory contradicts previous assumptions of SRB: that an equal number of X- and Y- chromosomes are produced, that neither has an advantage in fertilization, and that SRB differences are due solely to the male-female difference in prenatal mortality (James 1996). The evidence for the hormonal environment theory may be found in highly biased SRBs associated with gonadotropins and other reproductive hormones operating in the parents at the time of conception. Gonadotropins are either the follicle-stimulating hormone (FSH) or the luteinizing hormone (LH) produced in the pituitary gland by both men and women. FSH causes ovarian follicles and their egg to mature; follicle cells in turn produce estrogen, which in turn promotes growth of the uterus to allow favorable embryo implantation and which also stimulates the cervix to produce the mucus through which spermatozoa must pass to fertilize the ovum. In males, FSH is critical in the maturation of sperm through action on the Sertoli cells. In both sexes, LH causes gonads to secrete sex steroids. In females, LH is produced in a steady but low volume until ovulation, when a large surge is produced. The pulsing flows of LH may operate as a mechanism in sex determination (James 1987b).

Increased levels of gonadotropins in either parent may lower the SRB (James 1987). As I have noted earlier, gonadotropins have been positively correlated with plural births and with births of boys with cryptorchidism. Asians, who have relatively higher SRBs than white and blacks, also have lower levels of gonadotropins

and lower twinning rates (Soma 1975, James 1987b,), while blacks have higher levels of gonadotropins and higher twinning rates than do other groups (James 1987b, Kulch et al. 1993). Based on examination of bi-racial couples and other factors, it appears that the race of the mother is significant to the higher twinning observed in black births, which offers strong support that maternal, not paternal, factors affect twinning rates in general (Khoury et al. 1986); if such is true, maternal factors might also be more significant in sex determination.

A satisfactory explanation for these differences among racial groups has not been found. The difference may be innate or genetic, as suggested by Visaria (1967) in his review of over 70 populations with mostly complete birth registrations, or the difference may result from differing hormonal responses to social or environmental conditions, or it may be a combination of both. James (1987b) suggested that higher gonadotropin levels could result from subordinate positions in society or lower socioeconomic status. Freeman (1934) found racial differences in the weight of the pituitary gland, with blacks having the heaviest (800 mg), whites, an intermediate value (700 mg), and Asians, the lightest (600 mg). To this evidence one may also add a growing medical literature examining the differential operation of the hypothalamus-pituitary-adrenal axis among races, reflecting a genetic and endocrinal approach to understanding why patterns of hypertension and other heart diseases, breast and prostate cancer, obesity, and other diseases differ among racial or ethnic groups. In the current state of SRB research, race is a convenient label for a combination of alleles that appear to have distinct responses to social and environment conditions.

Other research in hormonal environment suggests that parental levels of testosterone and estrogen at the time of conception, perhaps in combination with gonadotropins, also significantly influence SRB. Men with high amounts of gonadotropins and low levels of testosterone produce a significantly high number of girls (James 1996). High levels of testosterone in either parent will raise the SRB (James 1996b, Manning et al. 1996, Singh and Zambarano 1997). Manning et al. (2002) found, for example, that a high 2D:4D ratio (the ratio of length of the second finger to the fourth finger—a proxy for testosterone levels at the time of gestation) also correlated to higher

SRB; that is, more boys were born to those with higher testosterone signals. Similarly, the waist-to-hip ratio, another proxy for testosterone levels in both men and women, signaled a significantly high SRB for women presumed to have high testosterone levels. There is strong evidence linking high testosterone levels with testicular and prostate cancer (e.g., Bosland 1988) and men undergoing surgery for removal of cancerous prostates have relatively more sons (James 1990b). Prostate cancer is 35 percent higher in U.S. blacks than U.S. whites (CDC 2003); some studies have found higher testosterone levels in U.S. black males than in U.S. white males (Winters et al. 2001). While this would indicate that the SRB in black births should be higher as a result of higher testosterone in fathers, it appears under this extended hypothesis that the higher gonadotropin levels in black mothers must be the more dominant agent in hormonal environment influence.

If the parental hormonal environment does affect SRB, it follows that compounds or conditions that change the genital chemistry of parents will also influence the SRB. Gonadotropins used in *in vitro* induction of ovulation, for example, appear to result in a lower SRB (Gray 1991). Parents who desire to have a female child can increase their chances by treatment with the ovulatory drug clomiphene citrate prior to artificial insemination (Silverman et al. 2002). Men treated with methyltestosterone and gonadotropins produce an excess of sons (Sas and Szollosi 1980). James (1987a) estimated that the SRBs in groups treated by hormonal induction of ovulation were .05 lower than nontreated groups, a difference near to that observed among races and much greater than for range differences for season of birth, parental age, and birth order.

Exposure to lead will change the gonadotropin/testosterone levels in men and cause them to sire an excess of daughters (Dickinson and Parker 1994). As an occupation, lead industry workers and professional bus drivers are exposed to high levels of lead and have fewer sons than average (Lin et al. 1996). Exhibiting possible genotoxic effects, the SRB of men who set consumable carbon anodes in aluminum plants in Washington State was .381 (Milham 1993), suggesting that strong static and extremely low-frequency electromagnetic fields might depress SRB. Another potential agent of hormone disruption, X-ray exposure, may lower the SRB. Hama et al. (2001) found that

male radiologists have a lower SRB, and the more highly irradiated of this group have the lowest sex ratio among radiologists studied.

Endocrine disruptors and SRB

The line of research that motivated this thesis, the association between environmental toxins and lower SRBs (raised by e.g., Moller 1996, Allen et al. 1997, Davis et al. 1998), is based on the idea that disruption of the reproductive hormonal environment by these agents in residents of industrial countries might have corrupted the hypothesized normal hormonal control of SRB. The U.S. EPA and similar health regulatory agencies in other countries have been monitoring the concentrations and reported effects of compounds that may disrupt the normal functioning of the human endocrine system. A number of terms have been applied to these compounds and their subcategories. The National Academy of Science screens these studies under the term "hormonally active agents;" however, I will use the more common "endocrine disruptors." Endocrine disruptors may alter hormone functions in a number of different ways. Those with chemical structures similar to estrogens, androgens, or related hormones may bind to the intended cell receptors or otherwise mimic hormonal chemicals in the normal cell signaling pathway. Conversely, anti-estrogens or anti-androgens may block cell receptors or cell signaling pathways. Other substances appear to alter or confound the production or breakdown of natural hormones or the function of their receptors. Categorically, environmental endocrine disruptors include biocides, insecticides, herbicides, nematocides, fungicides, industrial chemicals such as solvents and plasticizers, certain heavy metals (especially lead, cadmium, mercury), PCBs (i.e., specific PCB isomers), and various compounds that are a degradation product of or impurity in other chemicals.

Wildlife studies of disrupted reproduction, deformed reproductive systems, or aberrant sexual behavior have focused on certain pesticides, polychlorinated biphenyls (PCBs), and polychlorodibenzo-p-dioxins (e.g., 2,3,7,8-TCDD) and other dioxins. Many of the suspect compounds bioaccumulate in affected species and thus expose embryos. According the Centers for Disease Control and Prevention (CDC), Atlanta, the average U.S. resident has accumulated several of these suspect compounds, along with hundreds

of others, in his or her fat tissues from food and environmental exposure. A number of studies have examined the effects on human SRB from occupational exposure to pesticides that have suspected endocrine-disrupting effects. Workers applying dibromochloropropane (DBCP), a fungicide, have markedly reduced sperm count and a strong tendency to produce female offspring (Potashnik et al. 1984). Moller (1996) found that men in the Netherlands exposed to pesticides in the workplace from 1978 to 1990 fathered an extremely low proportion of boys (.248), with those men believed to have higher exposures also producing the fewest males. Garry et al. (2002) found that the SRB of children born to fungicide applicators in the Red River Valley of Minnesota is very low.

PCBs and dioxins are perhaps the environmental toxins most suspected to have an estrogenic mimicry that might depress SRB in children of exposed parents. Egeland et al. (1994) found that men exposed to dioxin have large amounts of gonadotropins and low volumes of testosterone, a combination suspected to reduce SRB. Several SRB studies have examined the 1976 Seveso, Italy chemical plant explosion because of the high numbers of people exposed and the elevated dioxin blood levels in the exposed population. Of the 74 children born to the most highly exposed adults from 1977 to 1984, the SRB was only .350; of these, the nine sets of parents with the highest levels of dioxin produced no boys (Mocarelli et al. 1996). Ryan et al. (2002) found that children of pesticide workers working with products high in dioxin, such as 2,4,5-trichlorophenoxy acetic acid (2,4,5-T), had an SRB of 0.4. Of significant chemical exposures, the SRB-depressing effects of dioxin appear the most convincing.

Studies relating PCB exposure to SRB are not as compelling as dioxin associations. Weisskopf et al. (2003) found that mothers in the Great Lakes region of the United States during the period 1970–1995 had a lower SRB if their blood serum PCB levels were elevated. These authors found no association with paternal exposure to PCBs; however, Karmaus et al. (2003) found a higher SRB in the children of fathers with higher PCB blood serum levels. In the case of the largest documented PCB exposure of a population, no significant alteration of SRB was found in children born to Taiwanese women exposed in 1979 to PCB-contaminated cooking oils (Rogen et al. 1999).

Some authors assert that an association between lower SRB and environmental toxins is worthy of further study because of increasing evidence of general disturbances of the reproductive system observed among populations in industrialized countries (e.g., Davis et al. 1998). For example, the rate of hypospadias, a birth defect in which the male urinary canal is open on the underside of the penis, has doubled in all regions of the U.S. from 1970 to 1993. Also, the rate of surgery to correct cryptorchidism appears to have increased 2- to 3-fold during the past 30 years, and the rate of testicular cancer in industrial countries has reportedly increased by at least three times in the last 20 years.

The effects of environmental toxins on women's reproductive health may be harder to detect because of the higher natural fluctuations in their hormone levels. Hermann-Giddens et al. (1998) found that development of puberty in white girls is 6 to 12 months earlier and in African American girls 12 to 18 months earlier than reported in previous studies, an effect possibly attributable to environmental exposure to estrogen-like chemicals. Such chemicals might also affect the reproductive capacity of adult women. Several identified endocrine-disrupting chemicals are pharmaceuticals, such as diethylstilbestrol (DES), a drug given to pregnant women from 1948 to 1972 to help prevent miscarriages. Daughters of these women had an extremely high rate of a rare cancer at maturation (Crisp et al. 1998). To date, this remains the only widely significant health impact to humans known to be caused by an endocrine disrupting substance (Crisp et al. 1998), but it suggests that widespread human endocrine system effects from exposure to chemical substances are at least theoretically possible.

Sperm production and quality are among the most monitored signals of reproductive health, and endocrine disruptors have been implicated in recent studies that detected widespread changes. In some studies, the rate of sperm production in the average male in western countries was found to have decreased significantly in recent years, although this remains controversial. One of the most publicized of these studies found a decline of more than 40 percent in an analysis of 61 sperm count studies from laboratories worldwide (Sharpe and Skakkebaek 1993). Many researchers in this field have speculated that the cause of this decline is due to an environmental factor that disrupts the human endocrine system. Swan et al. (1997) concluded that the

preponderance of sperm-count and quality studies to date represents a significant decline in sperm count in the United States and Europe that cannot be explained by individual characteristics of the men tested and therefore must be the result of geographically distinct environmental conditions. Swan et al. (2003) inferred from carefully controlled sperm counts in Columbia, Missouri, located in a predominately rural area, and in New York City, Los Angeles, and Minneapolis, that sperm count concentrations may be lower in agricultural and semirural areas than in less agriculturally exposed areas. The possible presence of geographic variation in sperm quality parameters justifies exploration of geographic variation in SRB and other reproductive system effects.

Geographic distinctions in SRB and reproductive system effects

In seeking evidence of environmental toxin effects, some researchers have tried to identify the geography of this exposure at a more precise level than "industrialized nation." One might, for example, hypothesize that high exposure to endocrine disrupting chemicals is more characteristic of persons in agricultural areas where application of pesticides is common. In some agricultural areas, increased rates of birth defects have been seen in the general population. Geographic analysis of cryptorchidism in different regions of the Spanish province of Granada found that the rates of surgical correction for this birth deformity were 2.32 times higher in districts with high pesticide use (Rueda-Domingo et al. 2001). Allen et al. (1997) found an east-to-west gradient in the decline of sex ratio in Canada and noted the higher rates of decline were in provinces with a high use of pesticides. As these and other researchers into potential effects of endocrine disruption suggest, geographic monitoring of changes in sex ratio could prove a useful tool for assessing whether specific, avoidable environmental exposures are occurring in specific regions.

A number of researchers have tried to link occupational exposure to pesticides and industrial pollution emissions or effluents to more general exposures in the population. Olival et al. (2001) found that men exposed to pesticides in the most concentrated agricultural region of Argentina had higher estrogen concentrations and lower LH gonadotropin concentrations than nonexposed men. However, in Italy the SRB of firstborns was not different in areas with high concentrations of pesticide application

or industrial pollution in the period 1989–1993 (Figa-Talamanca et al. 2003). These authors did find a slight increase in the sex ratio from the north to the south of Italy and a lower SRB in the major urban areas. Astolfi and Zonta (1999a) also found negative trends in SRB in the major metropolitan areas of Italy compared to the rest of the country in 1970–1995; they also noted that the stillbirth rate was significantly higher in the cities. Williams et al. (1992) at first found an association between industrial air pollution and lower SRB in Scottish cities, but later retracted these results. The trend in this small number of studies seems to indicate, however, a slightly lower SRB in cities than in less urban areas.

Interest in this geographic distinction in SRB between urban and rural areas precedes concern about environmental toxins. Winston (1931) for example, reported higher SRBs for rural whites and blacks than for their urban counterparts. The distinction between rural and urban areas in China and India, with SRBs elevated by elective abortion, shows a high SRB in rural areas due to cultural preferences. However, James (1987a) in a comprehensive summary of sex ratio research found no compelling evidence that higher SRBs were characteristic of either urban or rural areas. However, he did find that the rate of dizygotic twinning has dropped significantly in urban populations worldwide, with a drop in Poland, a highly polluted country, of as much as 50 percent. Dizygotic twinning, as we have already seen, is associated with high levels of gonadotropins and could be a signal that higher SRBs might be found in urban areas than rural areas. However, this speculation counters the somewhat tentative findings of lower SRBs in metropolitan areas cited above. The study I propose in this thesis will examine this question more closely.

On a broader scale, latitude has been examined specifically as an SRB variable, with temperature and photoperiod differences as one hypothesis for observed variations. Like season of birth, latitude appears to affect SRB according to principles not as yet determined. Grech et al. (2002b) found that the SRB decreases in Europe as geographic latitude increases. Examining births from 1890 to 1995 in Malta and from 1990 to 1995 in Western Europe, they found that SRB differences were notable in three general latitude bands: Scandinavia, Central Europe, and the Northern Mediterranean. They also found

that the SRB is higher in southern Europe than in northern Europe, with three latitudinal bands dividing southern European, central European, and Nordic countries. These differences may be the result of genetic distinctions in the populations of these countries similar to the possibly innate differences between black and white populations in the U.S. The higher SRB in southern European countries may also reflect a hormonal mechanism that considers births in countries like Greece and Spain more hospitable to vulnerable males in more months of the year than births in more northern countries.

However, Grech et al. (2002a) found an opposite latitudinal effect in North America during the period 1958–1997: the lowest SRB in Mexico; a higher one in the U.S., and the highest in Canada. The SRB of the U.S was significantly lower than that of central and southern Europe, with which it shares latitude. Canada, arguably a less hospitable climate than the U.S. or Mexico, had the highest SRB. However, the study did not control for the much more diverse racial and ethnic makeup of the U.S. and Mexico compared to Europe, whose population was almost entirely Caucasian until the late 1950s.

Sex allocation theory and SRB

As background to developing a hypothesis for the evidence of geographic distinctions in SRB hormonal control mechanisms, I will summarize some theories of evolutionary biology of how such mechanisms evolved. To begin, it is reasonable to ask the most basic question of this field of research: why is the sex ratio near parity? Why, indeed, are males needed? Nature's hermaphroditic or parthogenetic species are capable of reproducing without the great energy spent in sexual competition, courtship and mating that could otherwise be invested in food gathering and reproducing, and the ability of a single individual to populate a new area seems a significant evolutionary advantage. However, sexual reproduction seems to provide the most efficient method for creating and maintaining a large and flexible gene pool. Mutations produce new genes that are constantly available in new combinations to respond to environmental change or new predators (Crow 1994), and sexually reproducing species more easily accumulate beneficial gene combinations (Muller 1964). New, potentially advantageous genes can be rehearsed in multiple genetic combinations in sexual species (Fisher 1930, 1958).

Fisher (1930, 1958) was the first to suggest why natural selection demanded a 1:1 sex ratio in certain species, particularly in birds and mammals. Using economic terms, he described sex allocation as parental investment for a stake in the genetic makeup of the next generation. In large populations, mating is random and the parental care investment from each mate in raising a male or a female is approximately equal. Male and female offspring receive, on the whole, equal amounts of genetic material from their parents. A parent's genetic material can be carried on to the next generation on an allele with either an X or Y chromosome so, in Fisherian terms, either a boy or girl is equally profitable as a gene carrier. The potential for reproductive success of either a male or female child cannot be predicted by the parents, so the reproductive profits, in Fisher's terminology, are the same from raising a boy or girl—reproductive profits commonly expressed as the number of grandchildren. Individuals that produce an excess of one sex would suffer fitness problems in the next generation as their offspring suffered increased competition. Inequalities will therefore stabilize to a 1:1 ratio. Fisher's model is a type termed *frequency dependent allocation*, defining the fitness of a genotype by its frequency—the excess frequency of one sex or another exhibits negative fitness. This adaptive mechanism must have evolved from a facultative ability in humans to manipulate offspring SRB (Werren and Charnov 1978). The current slight excess in males results from their slightly greater mortality during gestation, birth and nurturing. As such, their average cost over the whole population is less than that of girls, so they are produced in a slightly greater quantity. This equal-cost assumption contributes greatly to the elegance of Fisher's general model, but is not frequently employed in sex allocation theory developed since Fisher.

Many of the sex allocation corollaries and extensions to Fisher have been successful in modeling species other than birds or mammals, in which parental manipulation of offspring is considered by many researchers as limited and perhaps nonexistent. A frequency dependent selection model similar to Fisher's and worth considering for human populations is the *homeostatic* model, which predicts that parents may most profitably invest their resources in producing a child of the sex that moves the

local population closer to equilibrium. If females are in short supply, for example, parents have a greater potential for reproductive profits if they produce a girl.

Another facultative, adaptive model, and one of the most investigated theories in evolutionary biology, is that proposed by Trivers and Willard (1973), briefly introduced in my review earlier in a discussion of seasonal breeding. Trivers and Willard (1973) theorized that a mother can facultatively adjust her offspring's sex ratio to maximize her success in passing on her genes. Mothers in good condition will produce offspring in better condition than will mothers in poor condition. The level of condition is more important to males because a good-condition male will exclude other males from successfully reproducing and will impregnate more females himself. Therefore a female in good condition will have more genetic success by producing male offspring. Mothers in poor condition will maximize their reproductive success by producing daughters, who will have potentially fewer total offspring than a male, but a greater chance of reproductive success than would a male in poor condition. In short, natural selection in this situation drives the SRB to favor the sex that will have the greatest breeding success given its condition. Myers (1978) extended the Trivers-Willard idea of adaptive sex ratio variation by suggesting that the mechanism for allocation was preconceptual, not postconceptual as Trivers and Willard suggested, because postconceptual fetal wastage is an inefficient process not likely to have been created by natural selection. This assumes that quality and not quantity is selected for in human offspring, and that the costs of replacement of an aborted fetus are high. However, if sex selection occurs very soon after conception, the costs associated with replacing the conception may not be that high. Facultative control by the mother to adjust sex of offspring may occur in the early period of pregnancy or even preconceptually as the mother "reads" her environment and assesses whether a male or female represents the best investment given her condition and the resources available.

While it is difficult to generalize the complex field of sex allocation theory, it might be safe to say the Fisherian principles operate at the population level whereas dimensions that demonstrate differential costs of investment between sexes, like the Trivers-Willard effect, operate at the family or local population level. The Trivers-

Willard effect has had some confirmation in studies of deer (e.g., Clutton-Brock et al. 1982) and other animal populations but evidence of its presence in human populations remains controversial. Indeed, the concept that parental hormonal mechanisms exist for a sociobiological purpose profoundly challenges Mendelian principles that random meiosis and fertilization will produce approximately equal numbers of males and females. Discovering conclusive evidence of such influences is a primary motive in many human SRB studies. If changes in the hormonal environment of the parents influence the sex ratio, the action of these hormones may in some way have evolved to operate facultatively to respond to environmental changes in order to maximize reproductive success.

In human populations, adaptive sex allocation mechanisms could manifest themselves in a number of ways. One would be adjustment of population based on inequalities in the adult sex ratio, as the homeostatic model predicts. Animal studies show that females raised in the absence of males will produce litters with higher SRBs. In humans among East African groups with polygynous marriages, the SRB of wives living together was .873 (Whiting 1993). Lummaa et al. (1998) found evidence of SRB adjustment to adult sex ratio, and, possibly, also a confirmation of the Trivers-Willard hypothesis, in a study of births recorded at 21 church parishes in preindustrial Finland (1775–1850). During this period the overall effect on SRB of the existing adult sex ratio of persons considered of reproductive age (15–50) was significant. In the majority of the 21 parishes studied, more sons were produced when males were rarer than females in the adult reproductive cohort. This suggests that mothers in these parishes facultatively adjusted the sex ratio of their offspring in response to the local reproductive adult sex ratio (RASR) in order to maximize the reproductive success of their progeny. Birth rate and, presumably, also population growth rate increased when the RASR approached equality. However, these authors observed that the strength of these patterns varied across parishes, suggesting that socioeconomic or environmental factors other than the RASR may also have also influenced the SRB.

James (2000) examined the possibility that a homeostatic or stabilizing movement towards an equal SRB exists in large populations during the last 50 years. He examined

the SRB at ten-year intervals and tested its relationship to the adult population cohort aged 15–44, chosen arbitrarily as the primary reproductive age range, to see if response in the SRB was evident as an adjustment to RASR. If such a response exists and its purpose is to keep the population at an approximately equal sex ratio, it should be present as a negative correlation between the SRB and the RASR. In 41 populations, primarily based on national boundaries, he found both positive and negative correlations, some of them significant. The lack of a significant pattern indicates that an adaptive response is not present in large populations. However, he did note a significant negative correlation coefficient between the RASR of U.S. whites and the white SRB, as well as a significant *positive* correlation between the RASR of U.S. blacks and the SRB of U.S. blacks. During the same period, there was a significant rise in the SRB of white births in Australia with a positive correlation to the RASR in that country.

The Trivers-Willard effect has also been studied in terms of parental investments after birth. Gaulin and Robbins (1991) found some confirmation of the Trivers-Willard effect through indications of birth weight, interbirth intervals, and lactational commitments in a study of 900 U.S. mothers. Koziel and Ulijaszek (2001) attempt to extend the Trivers-Willard effect to say that parents of high social and economic rank will bias their investment to sons, while parents in poor circumstances will bias their investment towards daughters. In a sample of Polish children examining extent of breastfeeding of first born children as an indicator of parental investment, they found weak support for the Trivers-Willard hypothesis. Where fathers were better educated, a greater proportion of first born boys were breastfed longer than girls, while the opposite was true in families of fathers with poor education.

The Trivers-Willard hypothesis has been employed by anthropologists and other social scientists to suggest that female mammals can facultatively manipulate the SRB of their offspring based on their status in society and access to resources in that society. Some research suggests that women who are more dominant appear to be more likely to produce sons. Dominance is a characteristic underpinned by testosterone. The intrauterine maternal environment provides for factors such as follicular testosterone that allow for the differential access of X- and Y-bearing spermatozoa (Grant 1994, 1996).

Some studies have found a social status SRB bias. Mealey and Mackey (1990) examined female status in the polygynous unions of 19th century Mormons, where status among men was defined by one of five ordinal positions of church rank. These positions correlated highly to socioeconomic status. Birth documentation is also superior. They found that women married to men of high social rank produced more sons, while wives of those of low rank produced more daughters. While overall SRB was .519 [108], the SRB of women married to men in the highest rank at the time of birth was .684 [216.7], while those in the lowest rank was .512 [104.8]. When results were viewed by highest lifetime rank achieved by the husband, they had a similar pattern. However, there was not a stepwise pattern for the intervening three ranks. The wife-order status also showed that the higher order wives had more sons than the first wife. This could be not be correlated to husband's change of status or wealth. Perhaps later order wives represented females in better condition relative to those first married, as the Trivers-Willard hypothesis predicts. Later order wives may also have responded to the homeostatic cues of a large female household, as did the polygynous groups studied by Whiting (1993). In both the case of relative social rank and wife order, a threshold effect was observed in which significance could be observed at the extremes but not in interval steps.

No discernable SRB effect across parental occupations in modern populations has been found (Dickenson and Parker 1997, McDowell 1985), but biased SRBs can be found at the extremes of economic class. At least up to World War II, European royalty had a very high SRB of .578 (Norton 1940), while men in personal service—domestic servants, cleaners, barkeeps, waiters, barbers—have a lower than normal SRB (McDowell 1985). The children of U.S. Presidents have an SRB 50 percent higher than the rest of the population (Ridley 1994). The Trivers-Willard effect may also explain the earlier cited studies (e.g., Manning et al. 1997) that show men who marry women much younger than themselves will sire more sons—the "sugar daddy" effect—the ability of these older men to attract younger women being evidence of their power and wealth. Chacon-Puignau et al. (1996) perceived the Trivers-Willard effect in the fact that the highest SRB among the children born in the Venezuelan population 1988–1990 occurred

in those born to married and cohabiting couples and the lowest was for children of single women, who were at the lowest end of socioeconomic status.

Social and demographic changes

Perhaps the greatest difficulty in detecting evidence of facultative, adaptive effects in the human SRB is that most variations in SRB can have either adaptive or nonadaptive explanations. As Lazarus (2002) argues, for example, paternal age and birth order effects on SRB may be either physiological or psychological constraints. Alternatively, birth order might be considered adaptive where investments in first born sons were more beneficial than investments in sons born later in the birth order, such as in cultures in which first born sons receive the majority of family estates. Coital frequency might be the selected mechanism for adaptation favoring a greater number of offspring for younger fathers, with the SRB a nonadaptive byproduct. Also, at the population level, it is difficult to detect an adaptive mechanism when its purpose is to achieve the same result as that of random, Mendelian segregation: a 1:1 sex ratio. James (2000) suggests that the problem of detecting hormonal control mechanisms is compounded by the possibility of negative feedback that moves any biased SRB back to parity.

While acknowledging alternative explanations for observed biases, it is useful to review the social and demographic changes in the U.S. for the period since 1970 to determine if any of the factors I have reviewed are present in these changes and thus suggest possible causes for the decline in SRB during this period. These changes include child sex preferences and family size, marriage patterns, age of parents having children, racial composition, and technological controls of birth such as artificial insemination, oral contraception, and ovulation induction.

Child sex preferences may be changing in many cultures. A specific preference for one sex or another may be a characteristic of the cultures of some developing countries, but the current sex preference in developed countries may be a balanced number of boys and girls (Jacobsen et al. 1999a). This may be a preference by both parents for a boy and a girl, or the satisfaction of a slight preference for boys by the father and a slight preference for girls by the mother. Such preferences can be deduced from

what demographers call "stopping rules." Where couples prefer one sex, they will stop reproducing when a child of that sex arrives; when a balance is desired, the couple will cease reproducing when representatives of both sexes are in the family. Sex preference may therefore be deduced from family sex ratio, size, and fertility. So, for example, preference for sons can be deduced in Korean families, while preference for balanced sex ratios is seen in Danish families (Jacobsen et al. 1999a). In larger Danish families, however, a slight preference for daughters was deduced from the fact that couples with two sons were more likely to continue having children than were couples with two daughters.

A possible status-related effect of differential investment in gender in the U.S. was recently reported by Dahl and Moretti (2004), who observe that parental preferences for a child's sex have impacts on divorce, marriage, and child custody. In the U.S., parents with girls are more likely to be divorced than those with boys; divorced fathers are more likely to have custody of their sons than of their daughters; and unmarried women with daughters less likely to get married than those with boys. Unmarried mothers who have ultrasound tests that detect boys are more likely to engage in a "shotgun" marriage at the time of delivery. Families with two girls are more likely to have another child than those with two boys. However, it appears the preference for boys deduced from these data is waning in the U.S.; the divorce rate for couples with a single girl child was five percent greater than those with a single boy in 2000, down from an eight percent gap in the 1940s (Dahl and Moretti 2004).

Demographic changes in marriage, age of parents having children, child preferences, and family size should be examined when considering changes of the SRB within a population. Gutierrez-Adan et al. (2000) found that the recent decline in SRB in Spain correlated with the higher mean age of marriage and the older age at which women are giving birth; the delay of childbirth into the 30s is lowering the SRB by increasing maternal age and reducing family size. Such demographic changes have not been studied as a cause of the decline of SRB in the U.S., but similar patterns have also been observed here. Marriage and childbirth are delayed from earlier years as more women go to college and join the work force (U.S. Census 1993). Approximately 47 percent of

women were in the work force in 1990; women in all racial groups increased their participation in the workforce (U.S. Census 1993). In the same year over 63 percent of women in their early twenties had not married, compared to 36 percent in 1970. Birth rates for women aged 30–34 and 35–39 had the highest increase of any age group over the period 1970 to 1990, while births to women 20–24 had the least increase. The mean age for a woman having her first child was 21.4 in 1970; this had risen by 3.5 years in 1990 (U.S. Census 1993). Means for mothers in all racial and ethnic groups increased, reflecting both the rising birth rates for women in their thirties and forties and the relatively recent downturn in the teen birth rate (Matthews and Hamilton 2002). However, the actual age at first or subsequent births varies greatly by state and by race and Hispanic origin. In 2000 the average age of women at first birth ranged from a high of 27.8 years in Massachusetts to a low of 22.5 in Mississippi (Matthews and Hamilton 2002).

The older ages at which women are having children in the U.S. and a related increase in multiple births in the last two decades is another possible explanation of the decline in SRB among white births. Martin et al. (2003) reported that the twinning rate increased 65 percent between 1980 and 2000, with the most pronounced increases in non-Hispanic white women. The rate of triplets has also climbed sevenfold during this period, with white births the highest of all races and ethnicity categories. This dramatic rise is attributed mainly to the increase in the use of ovulation induction and assisted reproductive technologies such as *in vitro* fertilization. In a 1999 study of U.S. triplet births, 43 percent were the result of assisted reproduction technology, 38 percent the result of ovulation induction, and only 20 percent the result of natural conceptions (Martin and Park 1999). Older women are more likely to have multiple births, even without fertility treatments, and the combination of fertility treatments and general deferment of childbearing to later years has increased both the rate of multiple births and the age range in which they occur. The incidence of multiple births before the onset of fertility treatments used to be concentrated in the 35–39 age group. Currently, women over 45 have a very high rate of multiple births; in 2000, for example, 40 percent of births to women over 50 were multiple births (Martin et al. 2003).

As we have already observed, ovulation induction, fertility treatments using gonadotropins, multiple births, and higher maternal age are all associated with lower SRB. Allan et al. (1997), most of whom are obstetrician-gynecologists, discounted these fertility treatments as a significant explanation of the SRB decline they first described in Canada, primarily because SRB decreases appear to have begun before these treatments were widely used. On a regional basis, the authors also note, the highest male declines were noted in the Atlantic provinces, where these treatments were less prevalent than in other Canadian regions. However, Dodds and Armson (1997) argue that ovulation induction, by itself, may be a more significant factor that Allen and his colleagues admit. Examining the state of ovulation induction in Canada, they note that the fertility rate in Canada is currently seven or eight percent. A survey of infertile women in the U.S. found that 43 percent seek medical treatment and of that percent approximately 20 percent undergo ovulatory induction. Assuming a 25 percent success rate and the assumption that such use reduces the SRB to .460 among those treated, they applied these figures to Canadian births and calculated an annual reduction in the number of males in excess of what Allen et al. (1997) described as the national decline. A similar study of the decline in U.S. SRB, which is only half the rate of the Canadian decline during the 1970 to 1995 period (Allen et al. 1997), might suggest that hormonally induced ovulation could by itself account for the U.S. decline in SRB.

An estimated 8 to 10 million women were using oral contraceptives in 1970 and the pill was in widespread use in other developed countries as well at that time (Pettiti and Wingerd 1978), corresponding to the beginning of SRB declines reported in several of those countries. Studies of association between oral contraception and SRB have mixed results. SRB among women who took oral contraceptives has been found to: decrease in women who took them for over two years (Keseru et al.1974); increase in births to women where contraception had failed (Shiono et al. 1982); or not change at all in births to women who had formerly used them, regardless of how long they had taken them (De Jong 1977). In general, oral contraception has not been advanced as a significant factor in the chemistry of births, but it may operate to reduce family size, and, thus, lower SRB as a function of the birth order effect. In Chapter 1, I reported the

speculation of Ulizzi and Zonta (1995) that Italy's rising SRB during the period when SRBs were reported declining in many other European countries was perhaps the result of that country's relatively later adoption of birth control methods. I speculated that Roman Catholic Ireland's somewhat similar pattern was for the same reason. Oral contraception is a powerful means of facultative control that has allowed couples to exercise a form of sex selection by limiting family size after children of the desired gender are born or balance is achieved. Perhaps birth control and the reduced preference for boys observed in the U.S. (Dahl and Moretti 2004) has contributed to the decline in the U.S. SRB.

Elective abortion, a major facultative tool for sex selection in some countries, as we have seen, has not been seen as a factor in any countries where SRB has been seen to decline (Davis et al. 1998). Parazzini et al. (1998) speculated that because of the improvement in prenatal diagnostic techniques, the detection of abnormalities motivating abortion would affect the male proportion the hardest, since congenital diseases are more prevalent in males. However, these should not have been a factor before the early 1980s. Marcus et al. (1998) also considered the possibility that increases in induced abortions of first pregnancies from the period 1969 to 1995 had decreased the SRB among white births, reasoning that lower birth order pregnancies are more likely to be male. They looked for evidence that births in the later years of this period had a higher number of first births that were second pregnancies. Such births would be likely to have fewer males than first births from first pregnancies, due to the birth order effect. However, no evidence of this effect was found. It does not appear that elective abortion is a significant factor in the decline in U.S. SRB.

Improvements in obstetric care have resulted in a decrease of stillbirths worldwide (Kalter 1991) and could have significant impacts on SRB. The immediate impact of improved prenatal care would appear to be an increase in the male fraction of births, since males are more susceptible to mortality from poor prenatal care. Improvements in U.S. obstetric care might explain the increased SRB in black births, but is the decline in white SRB an indication of poorer obstetric care or the result of some mechanism of sex allocation?

Sex allocation theory says that as more males survive to reproductive age, the SRB will adjust to lower the number of males. As Ulizzi and Zonta (1993, 2001) observed, a reduction in the SRB should be observed as a result of improved survival of males. They found that the Italian and U.S. white populations have experienced improvements in the rates of stillbirth, mortality within the first month, and mortality within the first year of life. Recently, male-specific mortality has been decreasing in all age groups and is approaching identical values for both sexes. According to expected Fisherian adjustment, the SRB will be the same as the sex ratio at reproductive age if these trends continue. While this explanation seems to apply to the declining trends in U.S. births to white mothers, the increasing trend of black births is not explained by a presumed parallel increase in obstetrical care.

Changes in racial demographics might also be a factor in SRB trends. Khoury et al. (1984) found that the SRB of births to interracial black-white couples (.511 [104.6]) was between the SRB for two black parents (.508 [103.3]) and the SRB of two white parents (.514 [105.9]). Increases in the number of interracial marriages since 1970 might therefore be a factor in the U.S. SRB decline. In 1970, only 0.6 percent of married couples were classified by the U.S. Census Bureau as mixed race; by 1995, the percent had increased by four times to 2.5 percent. The percent of black/white marriages has increased by approximately the same proportion: 0.14 percent to 0.57 percent, with the ratio of black husband/white wife marriages slightly increasing from 58.5 percent to 59.2 percent. Marcus et al. (1998) explored the potential for racial mixing to account for the increase in black SRB and the decrease in white SRB since 1969, based on the increase of births to interracial couples from 1 percent in 1960 to 4 percent in 1992. However, they found that the adjusted increase in the logistic regression odds ratio for male births (1.02) was smaller in magnitude than the decrease in odds ratio for male births to white mothers (0.994). Changes in the SRB of the U.S. could also be plausibly explored as demographic changes in the number of persons of Hispanic origin. The NCHS has tabulated SRB values by Hispanic and non-Hispanic origin for both white and black U.S. populations for a number of years. However, changing racial and ethnic definitions by

NCHS and the U.S. Census and inconsistent reporting success would make a long-term nationwide study of this potential effect difficult.

I have already reviewed socioeconomic attainment as a status factor in Trivers-Willard effects on individual family SRB. Socioeconomic decline in a population has been advanced as population stressor resulting in lowered SRB by at least one author (Catalano 2003). He found that the extremely high unemployment, high inflation, and a 50 percent drop in industrial production in Germany in 1991 resulted in the lowest SRB .509 [104] in that country since World War II. In the U.S., there have been no nationwide socioeconomic events of a similar catastrophic nature since 1970, although the collapse of the oil industry in Texas, evaporation of heavy industrial production in the Northeast, dissipation of rural economies in the Midwest, and other geographically specific socioeconomic declines might be explored for their effect on SRB. Nationally, there has been a decline in the number of black persons living below poverty level from about 31 percent in 1970 to about 22 percent in 2001; however, this level rose to about 35 percent in the early 1980s. Correlation between poverty level statistics and SRB may reveal socioeconomic stresses that change over time.

Since 1970, a quadrupling of the number of divorced persons, increases in immigrant population, greater diversity in racial and ethnic composition, a reduction in geographic mobility, and greater socioeconomic stratification, including access to health care (U.S. Census 2003b), are all sociodemographic factors potentially affecting the SRB. The rich trove of SRB literature could be also mined to form many additional theories of SRB decline. A 1993 study surmising that the increase in double-income couples had led to decreased sexual activity among such couples (dubbed DINS for Double Income No Sex) generated interest in a theory that the U.S. SRB decline could be attributed to a national disinterest in sex by these exhausted couples. I am tempted to indulge myself in further speculation in this vein, but will instead merely conclude that social and demographic factors cannot be excluded from any examination of changes in U.S. SRB. Postulated attributions of SRB declines in developed countries to environmental toxins have not sufficiently considered such factors. Martuzzi et al. (2001) suggested after a study of 35 million births that the varying SRB in European countries could perhaps be

better understood as concentrations of such cultural, biological, and economic factors than as results of environmental toxins' influence.

Summary of factors and the hypothesis

From a study of the literature of SRB, one can only deduce safely that there are many factors that influence sex ratio, so many that, in the musings of one researcher, it may soon be simpler to list factors that do *not* affect the sex ratio at birth. At the population level, race is the most consistently observed factor causing variation in SRB. Distinctions among U.S. populations of persons with Asian, American Indian, Hispanic, and other racial or ethnic origins are noted annually in reports by the NCHS. Low national SRB in many African countries may be attributed also to this factor, although the poor quality of vital statistics in those countries leaves this a still unresolved question. Currently, the percentage deviation between black and white populations is only 0.6 percent in the U.S., a gap that has been narrowing over the last half century (NCHS 2003b). The question of whether racial distinctions in SRB are innate, a result of different social positions, or due to some other factor is not resolved.

While controlling for race, the geographic description of U.S. SRB distribution I presented in Chapter 1 omitted many factors demonstrated to influence SRB. Family factors such as birth order, paternal age, plurality, and season of birth have been assessed in dozens of literature studies. While consensus has not been reached on the relative contributions or interrelationships of these factors, there appears general agreement that increased birth order and plurality consistently lower SRB, with slightly less support that increases in paternal age and, somewhat less still, increases in maternal age decrease SRB. Differences in these factors among racial groups may also exist. Seasonal variation in SRB has been observed, but may be obscured by modern living and working patterns. One consensus conclusion in SRB family factors study is the extremely small size of the variation in SRB in the general population that can be attributed to any suspect factor. Erickson (1976) found that the U.S. SRB changes by only two percent over the entire range of birth order values and that it accounts for only about 10 percent of the total variation in SRB.

The general conclusion of SRB studies is that when conditions are below optimal, fewer males are born. James' (1986, 1987b) theory that the hormonal environment of the parents at the time of conception influences SRB suggests a biological mechanism for many of the factors hypothesized to affect SRB, including birth order, parental age, plurality, seasonality, exposure to environmental toxins, hormonal induction, psychological stress, and smoking. Differing hormonal environments among races may explain their distinct SRB patterns. The changing levels of endocrinal compounds in the hormonal environment may have evolved as an instrument of adaptive control used by parents to increase their reproductive success in subsequent generations. Sex allocation theory provides an evolutionary explanation for the presence of hormonal control, while attempting to explain how such control may continue to operate in modern human populations. Adaptive mechanisms have been explored in both sperm production in the male and in the differential success of sperm in the uterus, but the whether the male or female has dominant control of the SRB in human populations has not been worked out in sex allocation theory. Grant (1994) presents evidence that mothers with more dominant or emotionally independent personalities produce more males, suggesting that female control of the offspring sex ratio is more significant than male control.

The influence of environmental endocrine disruptors on SRB at the population level remains unproven, despite the concentration of studies on this topic since the mid-1990s. If such a disruption exists at the population level, it has not yet been demonstrated, and the hypothesis is considerably weakened by the differential SRB trends among black and white populations in the U.S. Social and demographic factors have not been well addressed in studies of the decline in SRB in developed nations. The U.S. shift toward later childbearing years over the last thirty years and the increasing use of hormonally induced ovulation and other assisted reproduction technologies may be among the most significant of these ignored factors.

The literature of the human SRB is not without geographic perspective. Chambliss (1949) undertook to examine the SRB at the state level, but the poor quality of birth registration led him to dismiss any serious analysis of variation among states. Recent research to determine the cause of the SRB decline in industrialized countries has

also considered geographic factors. Studies in the U.S. and Europe (Grech et al. 2002a) have discovered latitudinal patterns of SRB variation. James has noted the differences in seasonality between the U.S. and Europe (1987a, 1990a), the seasonality of the SRB in black populations in the U.S. compared to other black populations (1987a), and the association of the SRB with the reproductive adult sex ratio in 41 mainly Caucasian populations (2000). The SRB of many industrialized nations has been evaluated to determine trends of decline, with some authors seeking to evaluate distinctions between urban and rural areas within nations (e.g., Astolfi and Zonta 1999a). The decline in the U.S. and Canada has been described at the regional and provincial level (Allen et al. 1997, Marcus et al. 1998).

However, the SRB in the U.S. has not been subjected to a thorough geographic analysis at the scale of the state, county, or city, and the description of the decline at the regional level is not exhaustive. The location, timing, and sociodemographic circumstances of the U.S. SRB decline in white populations have not been described with a full consideration of suspected confounding factors. Further, geographic analysis has not been used in service of determining whether there is an adaptive quality to the SRB of the U.S. When James (2000) examined the SRB among national populations, he speculated that analysis of an adaptive mechanism such as response to the RASR might not be appropriate at such large geographic scope, since parental perception of the local RASR was more important than a national aggregate. The compelling nature of the results of Lummaa et al. (1998) in detecting SRB changes among church parishes in preindustrial Finland further supports the contention that evaluation of factors at local geographic levels is a fruitful technique for illuminating the nature of the SRB.

My literature survey has been broad, yet has ignored many interesting facets of SRB and sex allocation research. I have not discussed, for example, sex-allocation extensions such as mate-attraction theory, nor the implications of fetus-controlled adaptive mechanisms that might compete with parental adaptations for sex determination. However, this survey does serve to demonstrate that research in SRB does not stand up to easy or simple assumptions. From its perspective, I return chastened to my original hypothesis of environmental toxin disruption of U.S. SRB and the preliminary analysis of

geographic differentiation. I have revised my hypothesis to consider environmental toxin disruption as only one of possibly several hormonally related influences of the SRB. I conclude from the literature that factors influencing the SRB are a combination of physiological constraints and both adaptive and nonadaptive consequences of social, demographic, and environmental influences, with parents exercising both facultative, conscious control and subrational ancestral facultative manipulation operating through hormonal pathways. Some disruptions of these hormonal pathways have been demonstrated in extreme exposures to certain chemical compounds, and modern living and working patterns may also misdirect or confound the operation of hormonal mechanisms evolved for ancestral environmental conditions. My revised hypothesis is that ancestral facultative adaptation continues to be a force in human SRB that may be detected at the geographic level of the U.S. county and state, but may be obscured in larger populations. In the following chapter I will present methods to test whether a number of conditions hypothesized to influence the SRB in adaptive ways could be detected at the geographic level of the U.S. county.

Chapter 3. Methodology

Present state of SRB population-based studies

The prospective investigator of SRB has many research threads from which to derive methods and models. Researchers continue to assess the potential impacts of endocrine disrupting chemicals on human SRB and other measures of reproductive health (e.g. Rogan and Ragan 2003, Weisskopf et al. 2003). Animal researchers are seeking to quantify SRB effects of environmental endocrine disruptors using mice (e.g., Tanaka 2003). Investigators continue to study cultural and religious influences on gender selection (Zietlin et al. 2002), and the impacts and determinants of the looming reproductive adult sex ratio crisis in India (Bandyopadhyay 2004, Bhat and Zavier 2003) and South Korea (Oum 2003). The effect of the psychological stresses of recent wars on SRB is being evaluated (Saadat and Ansari-Laril 2004, James 2003, Zorn et al. 2002). Preferences for male or female children in Nepal have been surveyed (Leone et al. 2003), as well as SRB and the seasonality of births in southwest Siberia (Melnikov and Grech 2003). Facultative manipulation of offspring sex ratio has been examined in primates (Brown and Silk 2002), ungulates (Sheldon 2004), mares (Cameron and Linklater 2002), house wrens (Albrecht 2002), fig wasps (Greef and Ferguson 1999), and many other animal studies of the Trivers-Willard effect that I have not reviewed in this thesis.

There also continue to be a number of studies of how SRB appears to function in human populations, a category into which this thesis falls. In Table 8, I review the topics and methods of these studies in the last five years, many of which investigated possible environmental toxin impacts. The implicit and sometimes explicit hypotheses of many of these studies relate to the discovery of underlying adaptive factors relating to SRB, but little progress has been made since 1993, when W.H. James advised:

> It is not yet possible to tell whether any given sex ratio bias is adaptive or the consequence of some physiological constraint…the problem should best be tackled by examining the proximate mechanisms underlying sex ratio variations which are likely to be far fewer than the number of variables with which sex ratio has been found to vary (James 1993).

The fact that many variables have been identified as significantly associated with the sex ratio may be a function of the substantial power associated with the use of large

Table 8. Summary of Methods for Recent Human SRB Population Studies.

Topic	Population setting	Controls	Methods	Authors
Declining SRB in Denmark	National vital statistics—Denmark: 1851–1995	None specified	Contingency tables using χ_2	Moller 1996
Declining SRB in Canada	All available births for Canada 1930–1990; U.S. 1970–1990	None specified; factors such as hormonal induction and immigration discussed in general terms	Logistic regression; national and division calculations	Allen et al. 1997
Correlation of SRB and adult reproductive sex ratio	21 parishes in Finland 1775–1850; 15 year intervals	None specified	Spearman rank correlation after sequential Bonferroni correction	Lummaa et al. 1998
Changing SRB in the United States	All NCHS recorded births in U.S. 1969–1995	Maternal age, paternal age, birth order, race	Logistic regression; separate models for white, black, other racial groups; year of birth added to detect significance of decline	Marcus et al. 1998
Trends in SRB in 29 countries	Live-birth male and female counts for World Health Organization database 1950–1994	Year of birth	Mean values stratified by three time periods. Observations of declines vs. constants, no statistical method specified.	Parazzini et al. 1998
Reduced births in Italian cities	National vital statistic records 1970–1995	Year; metropolitan vs. nonmetropolitan; region	Logistic regression; comparison of results to stillborn birth statistics by region	Astolfi and Zonta 1999
Natural SRB variation in Denmark births	All births from National vital statistics 1980–1993	Plurality, birth order, parental age, sex of preceding sibling	Logistic regression	Jacobsen et al. 1999
Comparison of SRB between heavily polluted town and nonpolluted town	1981–1983 births in Monchegorsk, Russia and Apatity, Russia	None specified	Kruskal-Wallis test of SRB	Kozlov 1999
SRB and environmental temperatures	National vital statistics—Germany 1946–1995	None specified	Spearman's rank coefficient between SRB by month and environmental temperature data lagged by ~10 months	Lerchl 1999
Decline in Japanese births	National vital statistics—Japan; 1970–1990	None specified	Linear regression of SRB values	Ohmi et al. 1999

Table 8. (Continued).

Topic	Population setting	Controls	Methods	Authors
SRB trends in 250 years of preindustrial Finland	Vital statistics and other sources for Various sources for 1751–1997	Father's age, mother's age, the age difference between husband and wife, and birth order of the child for most recent years	Various; primarily linear regression on 5 and 9 year running averages to test time trends	Vartainen et al. 1999
Secular movements in populations based on hypothesized homeostatic adjustment to adult sex ratio	41 national populations or national population subgroups at five 10-year intervals	Race population segregation	Spearman's rank coefficient for correlation between SRB and RASR of adults aged 15–44	James 2000
Impact of parental and biological variables on U.S. births	U.S. NCHS datasets; 1964–1998	Birth weight, race (white-nonwhite), father age, mother age (by five years intervals)	Logistic regression; data aggregated by group (birth weight, race, etc)	Nicholich et al. 2000
Declining trends of SRB in Europe	WHO national data for 23 countries in Europe 1950–1996	Year; country	Linear regression of SRB for 1st year and average for last three years	Martuzzi et al. 2001
Trends in SRB in Hungary	1950–1999	Maternal age, birth order, paternal age	Logistic regression	Orvos et al. 2001
Latitudinal differences in SRB in Europe vs. North America	WHO national data by year 1958–1995, North America; and 1950–1999 Europe	None specified; authors said that SRB influences were probably "multifactoral"	Contingency using χ_2 for trend, with confidence level using Fleiss equations	Grech et al. 2002
Economic stress hypothesis of two Germanies	SRB by year in East Germany and West Germany; 1946–1999	Year	Box interrupted time series test; autoregressive, integrated, moving average (ARIMA) modeling	Catalono 2003
Environmental factors (pesticides, industrial pollution, urban pollution) in Italian births 1989–1993	All Italian births during 1989–1993	Birth order and plurality (selected only first born singletons and maternal age	Standardized maternal age by region; calculated sex ratio percentage by region; Pearson's χ_2 test to compare crude male proportion values; Cochran's Z-test to standardize values	Figa-Talamanca et al. 2003

population sample sizes (Chahnazarian 1988). Reviewing these associations, it remains unclear what mechanisms might be operating to effect SRB biases, nor whether the independent variables examined are associated with other factors not controlled for in the study. As Table 8 shows, there are an uncomfortable number of SRB studies in which only one independent variable is studied. Given the extremely small variation of SRB in populations, control for suspected confounding effects is absolutely necessary to make a convincing case for the influence or lack of influence of the factor under study. As I summarized Lazarus's review of SRB studies in Chapter 1, lack of consideration of confounding influences, lack of understanding of the mediating influences on SRB (such as sperm X-Y chromosome ratios and timing of fertilization differential), and lack of an evolutionary basis for understanding SRB variation have been the major reasons we do not have a unified understanding of SRB (Lazarus 2002).

Sex allocation theory provides an evolutionary basis for approaching SRB study, but it has had a rocky road for empirical acceptance in human SRB research. One of the most hailed and successful branches of evolutionary biology, sex allocation theory has applied its elegant models successfully to some specialized species (e.g., haplodiploid insects), but has not been as assured in its modeling of bird and mammal species' adaptation. Individual studies show patterns of great promise but fall short as attempts are made to repeat them or as nonadaptive explanations are offered with better support. Clutton-Brock (1982) warns that "The very flexibility of adaptive arguments undermines their credibility since there are few trends to which a plausible adaptive explanation could not be fitted." (Note, however, that Clutton-Brock and his colleagues have contributed significantly to the study of Trivers-Willard effect in their classical studies of red deer populations on the Scottish isle of Rum (Clutton-Brock et al. 1984, 1986)).

Lazarus (2002) reviewed 54 analyses of human studies that examined the association of SRB and status measures such as social class, education, church rank, wealth, and entry in Who's Who, and found that approximately half (26) support the Trivers-Willard general hypothesis This and other findings may be consistent with, if not evidence for, the possibility that this mechanism exists in human SRB. Such research as the recent finding that thin women bear more daughters than their more robust sisters in

poor areas of Ethiopia (Gibson and Mace 2003) makes facultative adaptive SRB manipulation an attractive explanation for such effects in human SRB. Despite many efforts, however, proof of adaptive mechanisms in human SRB has been resolutely elusive. Cockburn et al. (2002) summarize the logical skein awaiting anyone seeking evidence of adaptation in SRB research results: 1) selection for sex-ratio manipulation could be weak or absent and Mendelian segregation of chromosomes determines the SRB of approximately .500; or 2) this adaptation is present, but unable to overcome the constraints of Mendelian segregation; or 3) this mechanism favors equal investments in males and females, which is achieved by Mendelian segregation; or 4) the effect of facultative adjustment is to create a sex ratio near parity, the same as Mendelian segregation. Finally, facultative, adaptive adjustment of sex ratio could be present, but modern social complexity leads to a variety of selection pressures operating independently of natural physical or biochemical constraints, so selection vectors are operating in many different directions and their net effect is zero, or a sex ratio at or near parity.

Not dissuaded from pursuing this line of evidence, I offer in my thesis, at the least, a previously untested geographic arena in which these speculations may play out. The era of geographically precise U.S. birth registration records begins at the same time as the suspected beginning of a U.S. decline in SRB. James's (2000) study of 41 national populations did not support Fisherian mechanisms in the relationship of SRB to adult sex ratio, but Lazarus (2002) argues that "since any effect of adult sex ratio must work through parental perceptions, the more local analysis of Finnish parishes [referring to Lummaa et al.(1998)] seems more appropriate." If parents' internal hormonal environments are influenced by, for example, the RASR, they are based on parental perceptions of the local RASR, not the measured national RASR. Analysis of these values at the state or national level is unlikely to reflect the visual, tactile, auditory and vomeronasal cues that daily living offers to fecund couples, as well as the general perceptions of the local mating market shared among members of the same community. The smaller geographic area of the U.S. county better serves this purpose, although I

recognize that the county frequently also aggregates the experiences of a large number of individuals with significantly different social and economic contexts.

For the period of my study, the 3,141 populations represented by U.S. counties or county equivalents (all henceforth collectively referred to as counties) are more numerous than any geographic units reviewed in previous SRB population-based studies and are geographically constant through three U.S. decadal census periods during which detailed demographic and economic variables have been recorded. These include measures of population density, racial composition, income and poverty rankings, employment, crime, age distribution, mortality, health, fertility, and other characteristics of living and working conditions that vary from county to county, creating differing hormonal responses in their residents. A number of these variables may serve as surrogates for hypothesized local stressors affecting the parental hormonal environment and evoking responses that have, or once had, an adaptive purpose.

Approach

Data

In Chapter 1, I discussed the details of the NCHS Natality Data Set I received by special request from NCHS. In brief, these data contain all field values for all births recorded by state vital statistics agencies and collected and published by NCHS for 1970, 1975, 1980, 1985, 1990, and 1995, with the addition of county-of-residence identifiers for each birth record. Among these fields are family factor variables that have been shown to influence the SRB, including:

- *Age of mother.* Hypothesized to decrease SRB with increasing age.
- *Age of father.* Hypothesized to decrease SRB with increasing age.
- *Birth order of child.* Hypothesized to decrease SRB with later position in birth order within a family.
- *Race of child.* Hypothesized to be higher for white births than for black births; SRB among other races also varies.
- *Plurality of birth* (after 1972 in NCHS dataset). Hypothesized to decrease SRB for twin and triplet births.

- *Date of birth* (from which season of birth may be extracted).

  Hypothesized to influence SRB resulting in more boys in warmer seasons.

These individual birth factors are useful in a statistical model that examines external environmental SRB influences by allowing control for innate biological characteristics that might bias a birth towards either gender. Some previous examples of using such controls are summarized in Table 8. The consistent influence of these factors on SRB may express some form of adaptive mechanism operating over the course of the reproductive life of a family, biasing, for example, the sex of children born to older parents versus younger parents. My interest in this thesis is to control for these variables in a search for external environmental SRB factors that might be detectable at the observation unit of the U.S. county. The county of residence recorded in natality data is assumed to be the environment of the mother and father of the child prior to and after conception of the recorded birth. In fact, the U.S. Census reports that 14 to 17 percent of the U.S population will move in a typical year. Approximately 60 percent of moves are within the same county (U.S. Census 2004), so 4 to 7 percent of parents may have lived in counties other than the county of residence recorded in birth statistics. However, I assume that this volume of movement will not significantly bias the results of any of the variables I have analyzed.

Such external factors may be extracted from socioeconomic data gathered by the U.S. census for 1970, 1980, and 1990. These data are published periodically as the City and County Data Book (CCDB) at approximately 10-year intervals (U.S. Census 1972, 1984b, 1994) and are available in electronic form from the Inter-university Consortium for Political and Social Research (ICPSR). Although data fields do not exactly correspond in all publication years, a number of data common to each census year represent social and demographic measures that can be used to broadly distinguish hypothesized external hormonal influences present in one county from those in another. In addition to the U.S. Census CCDB data, I also used age, sex, and race (ASR) data by county that has been compiled by the U.S. Census for these decadal years (U.S. Census 1984a, 1992) and distributed electronically by ICPSR.

Selected independent variables

Based on my review of the literature and of the data available in the CCDB publications, I selected a number of variables that might represent individually some selective pressure for facultative adaptive manipulation of the SRB, or some condition that disrupts the hormonal mechanism responding to such stressors. I term these selective pressures and conditions hypothesized socio-environmental hormonal stressors (SEHSs). They include:

Urban-rural environment. Accumulations in human tissue of environmental toxins similar in nature to endocrinal hormones may distort the normal function of adaptive hormonal control by biasing the internal hormonal environment of the parent toward production of females. As in Astolfi and Zonti (1999a), I will examine the SRB difference between urban and rural/farm counties. My analysis identifies counties with hypothesized high exposures of the general population to agricultural pesticides where I would expect to find lower SRBs. I recognize that inferences about any observed differences would be difficult to attribute to environmental toxins, since other aspects of the difference between rural and urban environments might also affect the internal hormonal environment. For example, urban dwellers might be characterized as more estranged from seasonal signals than rural residents. Urban residents are also exposed to higher concentrations of automobile emissions and industrial pollutants than rural residents. However, detection of a significant distinction between SRB influences in these two environments would be, whatever its cause, useful to forming a theory of SEHS influence.

Homeostatic adjustment. I will examine the influence on the SRB of the county RASR — defined, per Lummaa et al. (1998), as the ratio of reproductive males aged 15–50 divided by the total males and females aged 15–50. Based on the literature, logistic regression should show an inverse relationship between SRB and RASR as new births adjust to excesses of either sex in the adult population. I hypothesize that this inverse relationship will be present at the county level where prospective or newly impregnated mothers will respond to cues about local RASR. The actual county RASR will serve as the parentally perceived RASR. Lummaa et al. (1998) suggest that observed adjustments

in the Finnish population were adaptive, but James (2000) says stabilizing movements such as this would not necessarily be evidence of adaptive mechanisms. I have found no study of SRB homeostatic movements in the U.S. besides that in the James (2000) study of national black and white population SRB over the last 50 years.

Racial proximity stress. The reason for the different SRB of white and black populations in the U.S. is not known, although James (1987b) believed that blacks may have higher levels of gonadotropins than whites. This hormonal difference, if it exists, may be the result of ancestral adaptation with little connection to modern survival needs, such as the differences in adult lactose tolerance observed among racial groups. However, as James (1987b) speculated, minority status in society might affect the internal hormonal environment of a racial group and might thus explain the lower SRB of blacks in the U.S. and U.K. Such a mechanism, if it exists, would lead to fewer boys in the births to the minority population, according to adaptive theory. Even without an adaptive purpose, fewer boys might be born to minorities due to the psychological stresses of disadvantage. On a county level, the strength of this association may vary with the racial composition of the county. I will test a hypothesis that there is an inverse relationship between the SRB in either white or black births and the percentage of the total population of the other race.

Socioeconomic stress. I have reported evidence that stress may cause a reduction of the SRB (e.g., Lyster 1974, Fukuda 1998), but results associating low economic status with SRB are mixed. The notion that individual poverty could affect socioeconomic status is an old idea (e.g., Winston 1931) and some authors have associated higher SRB with higher socioeconomic classes (Teitelbaum 1970, Teitelbaum and Mantel 1971). Other authors have found no association (Erickson 1976, Rostron and James 1977). Studies associating SRB with local or national socioeconomic conditions rather than individual economic status are rare. I know only of Catalano (2003), who showed that macro socioeconomic stresses reduced SRB in the German population. I hypothesize that counties with lower socioeconomic status will have a lower SRB.

Statistical methodology

My primary statistical procedure is logistic regression modeling, chosen based on my review of recent SRB population studies (Table 8). According to Wilson and Hardy (2002), logistic regression modeling is a superior form of analysis for sex ratio statistics, although fewer than 30 percent of sex ratio studies reviewed by them (including studies of adult human sex ratio and animal studies) use this or similar generalized linear models. The remainder use nonparametric or classical parameter models. Citing the many advantages of logistic regression modeling in sex ratio research, Wilson and Hardy (2002) note that because the underlying error of the sex ratio is presumed to be binomial, this error is incorporated in the modeling process. Logistic regression modeling avoids the need to transform data to meet distribution requirements for parametric tests; it also has good power compared to many nonparametric tests. SRB researchers using logistic regression can thus avoid *ad hoc* transformations required by methods that must have normal distributions, or nonparametric tests that lack power.

An additional virtue of logistic regression is that it allows for the simultaneous testing of several interacting factors and covariates in a single model. I have prepared my dataset in a way that combines individual case variables, such as maternal age and birth order, with county social and environmental variables that are hypothesized to affect the hormonal environment — SEHSs. The goal of this approach is to allow me to create a multivariate model that considers both the biological condition of the parents and the local economic and social environment in which they presumably resided prior to conception and during gestation.

The binary logistic regression method contained in SPSS 12.0 (SPSS 2004) was used to test the strength of the association between the dichotomous dependent variable Y — the sex of the child — and selected SEHS variables, while controlling for the effects of family factor confounding variables. Using the logit model, this software uses standard nonlinear transformation of an ordinary linear regression to allow probabilities to fall between 0 and 1. For multiple independent variables, the logit equation can be given as

$$p(Y) = \frac{Exp(\alpha + \beta_1 x_1 + \beta_2 x_2 \ldots + \beta_i x_i)}{1 + Exp(\alpha + \beta_1 x_1 + \beta_2 x_2 \ldots + \beta_i x_i)} \quad (3.1)$$

Where, $p(Y)$ is the probability (p) that individual case i will be a member of Y, such that $p(Y)=1$ (in the case at hand, a boy), *Exp* is the exponential function raising the Euler number to the value within the parenthesis, α is the coefficient of the constant (i.e., the intercept, or the value of the independent variable x when Y is 0), and β is the coefficient of the independent variable.

SPSS creates the logistic regression model through use of an estimation procedure that maximizes the log likelihood, LL, for a model whose coefficient values are fit through iterative testing. SPSS reports the –2LL as the "model chi-square" to test significance of the logistic regression model. Model output parameters that I report in my results are:

1) the independent variable coefficient (*B*);

2) the standard error of the coefficient (*S.E.*);

3) the Wald statistic (*Wald*), the square of the coefficient divided by the standard error of the coefficient;

4) Significance (*Sig.*), reported as the probability value (p);

5) The exponent of the coefficient (*Exp(B)*), also termed the "odds ratio," or OR;

6) The 95 percent confidence interval (CI) of the odds ratio, calculated from the product of the SE * 1.96, then added or subtracted to the coefficient to get upper and lower bounds.

I used the OR as the most convenient term for describing the direction and strength of the relationship between the dependent variable and the modeled independent variables. In all models, I coded the dependent variable as 0 for girl, and 1 for boy, following the convention of the SRB expression. Independent variables in the model were either numeric, such as the age of the mother or the birth order of the child, or categorical (nonnumeric), such as residence within or outside of a metropolitan area, or a combination of both categorical and noncategorical individual variables. Categorical variables were "dummy coded" to number values for processing in the SPSS software.

SPSS allows the researcher to select a contrast method for categorical value coding. In all cases, I selected one member of the category to serve as what SPSS terms as the "indicator" category. The indicator category is not reported in the model output, but the coefficients of the other members of the category are reported in reference to this indicator variable. For categorical variables, SPSS also reports the Wald statistic and significance of the category as a whole, but ORs are only reported for individual category members.

For example, I selected the "White" category as the indicator member for the Race of Child category, which includes three members in one of the NCHS racial codings: White, Black, and Other. When the race of child category was included in a particular model, ORs were reported only for the Other and Black category members, which might both decrease the OR of the birth relative to the White indicator member. Therefore, the relationship among the members of the category must be interpreted in reference to the omitted reference member. My results specified the omitted member in each model that uses categorical independent variables.

SPSS also allows the off-setting of a cutpoint, or threshold, value from its default value of 0.5. Model-estimated probabilities above this number were assigned membership in the 1 (or boy) category; those below, to the girl category. In preliminary model testing I explored setting this value to the mean SRB value for the race and year of the sample (e.g., .512 for white births in 1990), but the test models did not have significantly different results from models run with the default of 0.5. Therefore, I left the threshold value at 0.5 in all final models.

The objective of using the logistic regression tool for this study is to create the most parsimonious model of independent variables that predict the outcome of individual birth cases. SPSS provides a number of options for model creation. Variables may be entered into the model in the order determined by the researcher (i.e., the "enter" method in SPSS). Logistic regression may also be used to test the fit of the model after each coefficient is added or deleted, called stepwise regression. SPSS provides a number of forward and backwards stepwise methods based on conditional, Wald statistic, and inclusion likelihood criteria. Stepwise regression is recommended for exploratory

modeling but is not recommended for theory testing (Menard 1995). Theory testing, in the context of this thesis, is the testing of my hypothesis that certain external social and environmental factors will bias the sex of the child in a manner consistent with a facultative, adaptive manipulation of the sex ratio by the parent. Backwards stepwise regression is considered useful for exploration, beginning with a fully saturated model and retesting the model after deletion of individual coefficients. This stepwise regression method is particularly useful where *suppressing* effects are suspected; that is, where an independent variable acts on the dependent variable through some relationship with a third independent variable. While providing some insights into the associations among variables, the results of stepwise regression testing are sometimes idiosyncratic and difficult to reproduce, so the researcher-selected and -entered method of model construction is generally preferred for theory testing. Therefore, I used this method to construct my final models, after initial testing with stepwise regression methods.

Detailed methods

Data preparation

The NCHS Natality Data Set, the CCDB dataset and the ASR dataset were imported into a standard relational database management software program and recoded. The primary recoding task for the NCHS Natality Data Set was to give each record a common Federal Information Processing System (FIPS) five-digit code for county locations; all NCHS county codes in the 1970 and 1980 sample years were converted to FIPS codes. For CCDB data, some data fields were created so each sample year contained the same measurement unit; for example, the "percent urban population" field had to be extracted from other data in the 1990 sample year but was already present in the 1970 and 1980 sample years. The ASR data were queried in each sample year to create county level RASR values for all racial categories, and for each race category.

To create the 1970, 1980, and 1990 County SRB Datasets (CSDs) used for final modeling, selected fields from the NCHS Natality Data Set for those years were merged with selected fields from the CCDB and ASR census datasets using the county FIPS code. The resulting three CSDs represent all births for their respective sample years except births whose county of residence is recorded as outside the U.S. In addition, I

elected to omit Alaska from the CSD dataset for several reasons. Alaska counties changed in name and number over the 1970 to 1990 period and NCHS county coding did not correspond to U.S. Census county coding for equivalent years. I considered including Alaska with aggregated state numbers, but some CCDB data fields could not be aggregated to state level numbers, particularly those with percentage values, and equivalent state data could not be found. Finally, including Alaska as a "county" would have violated the assumptions of geographic proximity I made in formulating some of my SEHSs.

CSD databases were imported into the SPSS software for final coding and modeling. I weighted the 1980 CSD to correct for sampling differences by NCHS. Because the NCHS 1970 dataset is 50-percent sampled in each location and the NCHS 1990 dataset is a 100 percent sample in each location, no weighting was applied to either year on the assumption that the 1970 50-percent sample would yield reasonably valid results across all geographic areas. However, approximately 10 percent of the 1980 dataset is 50-percent sampled, so I doubled the weight of all records in 50 sample areas in the 1980 CSD.

Preliminary model testing

Family factors testing. I conducted preliminary model testing on the 1970, 1980, and 1990 CSDs to confirm whether they produced results similar to those predicted by studies reported in the literature for family factor variables. If my results were similar, I could be confident that selected family factor variables could be used as control variables for my hypothesized SEHSs, as well as confirming that the data in my CSDs had similar characteristics to other populations studied. The results of my testing for family factor variables are reported in Chapter 4.

SEHS variable selection. I also did extensive logistic regression modeling on the 1990 CSD to select among many potential social and demographic variables from the CCDB and ASR data for final model testing. These included geographic residence categories such as:

- U.S. census regional division

- U.S. state
- Office of Management and Budget metropolitan designation
- Consolidated Metropolitan Statistical Area
- Population size class for the county
- Percent population change from the last census for county
- County population ranking
- County population per square mile
- County centroid latitude
- County centroid longitude

The county-level social, demographic and environmental variables I assessed included:

- Percent white population
- Percent black population
- Percent of population by age range
- Male population percentage
- Sex ratio of the population aged 15–50 (from ASR data)
- Birth rate to mothers under 20
- Deaths per 100,000
- Crimes per 100,000
- Civilian unemployment rate
- Median family income
- Median household income
- Median household income rank relative to all counties
- Percent families below poverty line
- Percent persons below poverty line
- Percent female heads of household
- Percent females in work force
- Percent agricultural workers
- Percent of county in cropland
- Percent farm population

- Percent rural population
- Percent urban population

I evaluated all these variables in the logistic regression model univariately with Sex of Child as the dependent variable; I also used various stepwise methods in combination with family factors and combinations with other social, economic and environmental factors. Stepwise regression methods were somewhat helpful for identifying variables of interest, but I found the results were highly sensitive to small changes in the independent variable set.

After this initial investigation, I selected a final set of county level SEHS variables to be evaluated in a series of multivariate logistic regression models for each year in the revised study period dataset. Selected SEHS variables were:

- Percent farm population
- Percent urban population
- Reproductive adult sex ratio (RASR)
- Percent black population
- Percent white population
- Percent families below poverty line
- Division of residence

As reported in Chapter 5, I analyzed SEHS variables in each CSD univariately and in models with family factors also included. Finally, I constructed a final model for CSD using a forward stepwise inclusion method to select among all SEHS variables for the best fit model.

Chapter 4. Logistic Regression of Family Factors

Individual family attributes that affect SRB—race, parental age, birth order, plurality—have been accepted to the point that they have reached virtual canonical status in the literature. James (1987a) speculated that the voluminous literature about this category of SRB influences might exist simply because, like Everest, the information is there (on the birth certificate). The real contribution of these family factor variables is not known and there is considerable debate on which factors actually bias SRB, how they may interrelate, and whether they have adaptive purposes. For example, a presumed inverse relationship between coital frequency and parental age might partly explain both the parental age effect and birth order effect. Adaptive purposes for these effects might be surmised, such as that early parental investments in sons pay off more in reproductive success than those for later sons, but cannot be supported conclusively by any research. Family-factor influences on SRB may be mediated by variable parental hormonal levels, possibly combining variable sperm sex-chromosome ratios in the father with cycle-timing mechanisms in the uterine environment. The physiological constraints of declining sperm quality in older men and older oocytes and wombs in women may contribute generally to a suboptimal environment for the more vulnerable male and thus a lower SRB.

With less acceptance in the literature, there is some evidence that the SRB varies by season, with some investigators theorizing that males are born when temperature and food availability is more optimal and more females when conditions are worse. A variation of the Trivers-Willard hypothesis supports seasonal breeding; males raised in poor conditions will have less breeding success when mature than will daughters raised in these conditions. However, even studies confirming seasonal variation in human birth quantities and SRB variation show wide geographic and temporal variability, diminished variation due to modern living and working patterns, and significant mitigation by socioeconomic factors. Matsuda and Kahyo (1994) found, for example, that the seasonality of marriages affected the seasonality of first-born children in Japan while environmental factors such as latitude and environmental temperature affected later born children. Temperature and photoperiod factors may influence SRB at the time of

conception or shortly thereafter, suggesting that this adaptive mechanism may be present in humans (Lerchl 1999, Cagnacci et al. 2003).

The lower SRB in twins and triplets is also well accepted (Martin and Park 1999), with the lower birthweight of multiple births again demonstrating a physiological constraint against male births. In general, multiple births seem an excellent adaptation, doubling, tripling, etc. the parental prospects for reproductive success, provided sufficient resources are available to bear and nurture extra children. Lummaa et al. (1998) showed that dizygotic twins on prosperous islands off the coast of Finland were far more numerous in populational terms than on the poorer mainland nearby. As twins must share intrauterine resources, they would in general be poorer in condition than singletons; Trivers-Willard would dictate that the SRB would generally be lower in multiple births. High levels of gonadotropins associated with multiple births might therefore be present as an agent of adaptive hormonal control.

There are other individual characteristics of parents that have been shown to depress the SRB (e.g., smoking) but they are incompletely reported in NCHS natality data over my study period. In any case, no multivariate regression analysis of SRB influences can ignore consideration of family factor variables. In this chapter I examine the significance of these variables in the 1970, 1980, and 1990 CSDs, and consider additional individual factors that warrant inclusion of a baseline model of confounding factors with a full model of SEHS variables.

My first set of logistic regression analyses evaluated child race, plurality, live-birth order, maternal age, and paternal age in each of the CSDs. Race of Child and Plurality were included as categorical variables. I selected the "white" member of the Race of Child category as the contrast variable, so logistic regression results show only "other" race and "black" race coefficients relative to the white-race category member. While the racial and ethnic subcategories of the other-race member have changed over the study period, this coding should allow control of the racial effect relative to other family factor variables. Plurality is coded as a categorical variable because multiple births higher than triplets are very rare and the association between SRB and plurality does not appear to be linear. The "singleton" multiple-birth category for each CSD was

used as the contrast member for the Plurality variable, except for the 1970 CSD because plurality was not recorded by NCHS until 1972. All other independent variables evaluated in the family factors analysis are numerical. Table 9 presents results for this analysis for all CSDs.

Results for each CSD showed consistent, significant contributions of race, plurality (for 1980 and 1990), and live-birth order to SRB. These variables were also significant in each CSD in univariate models when race is modeled separately. Maternal age and paternal age were not significant predictors of SRB in these multivariate models. However, in separate datasets for white and black births, paternal age was significant in univariate white-birth models for 1990 ($p=0.002$) and 1970 ($p<0.001$), but not for 1980 ($p=0.111$); and for black births in 1990 ($p<0.001$) and nearly significant in 1980 ($p=0.056$) and 1970 ($p=0.053$). Maternal age is significant for white births in 1970 ($p=0.014$) but not for 1980 ($p=0.100$) or 1990 ($p=0.295$). For black births, maternal age was significant in a univariate model in 1990 ($p=0.002$), 1980 ($p=0.036$) and 1970 ($p=0.007$).

In all cases these models confirmed the direction of the SRB bias generally found in previous studies. Relatively more girls are born to black mothers than to white mothers, at least in U.S. and European populations. First-born children are more likely to be male than later children. Singleton births are more likely to be male than twins or triplets. Younger parents also have more males, although this may be correlated to birth order. Regarding the relative influence of paternal age, maternal age, and birth order, my results are consistent with Erickson (1976), who found that neither paternal nor maternal age significantly influenced the SRB when birth order was controlled. The bulk of SRB demographic studies reviewed by Lazarus (2002) identified birth order as a significant variable, and in other studies than those finding a significant SRB influence in paternal or maternal age. Other recent studies, however, have found significant contributions to SRB from parental age; however, my goal in this thesis was not to decipher the relationship among these variables, but to control for their influence in geographic variable regressions. While paternal age appears to be a more significant SRB bias factor than

Table 9. Logistic Regression Results for Family Factors in All CSDs: Race, Plurality, Birth Order, and Parental Age. Bold indicates $p \leq .05$.

	B	S.E.	Wald	Sig.	Exp(B)	95.0% C.I.for EXP(B)	
						Lower	Upper
1970 CSD (N=1,683,365)							
Race of child			24.975	**.000**			
Black	−.024	.005	24.974	**.000**	.976	.967	.985
Other	−.003	.012	.079	.778	.997	.974	1.020
Live-birth order	−.004	.001	10.239	**.001**	.996	.994	.999
Father age	−.001	.000	2.972	.085	.999	.999	1.000
Mother age	.000	.000	.564	.453	1.000	.999	1.001
Constant	.075	.008	88.698	**.000**	1.078		
1980 CSD (N=3,150,379)							
Race of child			34.914	**.000**			
Black	−.022	.004	33.752	**.000**	.978	.971	.986
Other	−.010	.007	2.094	.148	.990	.977	1.003
Plurality			36.838	**.000**			
Twins	−.052	.009	36.384	**.000**	.949	.933	.965
>Twins	−.042	.059	.507	.476	.959	.853	1.077
Live-birth order	−.003	.001	8.145	**.004**	.997	.995	.999
Father age	.000	.000	.003	.960	1.000	.999	1.001
Mother age	.000	.000	.062	.840	1.000	.999	1.001
Constant	.066	.006	106.102	**.000**	1.068		
1990 CSD (N=3,460,794)							
Race of child			34.587	**.000**			
Black	−.019	.003	31.312	**.000**	.981	.975	.988
Other	.006	.005	1.791	.181	1.006	.997	1.016
Plurality			24.641	**.000**			
Twins	−.036	.007	24.157	**.000**	.965	.951	.975
Triplets	−.015	.038	.199	.656	1.018	.952	1.099
Quads	−.117	.148	.253	.615	1.079	.803	1.5449
Live-birth order	−.003	.001	12.425	**.000**	.997	.995	.999
Father age	.000	.000	3.380	.066	1.000	.999	1.000
Mother age	.000	.000	.072	.788	1.000	1.000	1.001
Constant	.073	.006	162.756	**.000**	1.075		

maternal age in my data, including paternal age in my models would greatly increase the volume of missing value records, as shown in Table 10.

When paternal age is removed from the analysis, the missing case proportion in all CSDs is considerably improved, dropping to 1.1 percent in 1970, 0.9 percent in 1980, and 0.6 percent in 1990. Inclusion of paternal age would substantially decrease the geographic extent and volume of the dataset, particularly for black births which may omit paternal age information for up to 45 percent of records in a sample year. Further, my models showed that the OR for either paternal age or maternal age is only slightly reduced from 1.00 (typically .999 or .998), and, as mentioned above, is not usually significant when birth order is included in the analysis. Therefore I judged that omitting parental age variables was not as detrimental to my thesis goal as the loss of geographic coverage would be if these variables were both included. I omitted both the paternal age and maternal age variable from the baseline family factor variable set, leaving only race, plurality, and live-birth order.

Using this revised variable set I evaluated the SRB effect of season of birth. Based on general theory that fewer boys will be born in the resource-poor fall and winter, I created a variable called Season and aggregated all births in March, April, May, June, July, and August as a Spring-Summer category member—the reference category—and remaining fall-winter months as the Season variable to be analyzed as the categorical variable. This simple categorization represents a crude distillation of the nuanced influence of climatic and weather influences, but will serve for the current model as a potential confounding factor. Results of the final family factors analysis are presented for each CSD in Table 11. These results showed highly significant results for race, birth order, and plurality, with the fall-winter Season factor significantly decreasing the SRB in 1970 only, but also nonsignificantly in the 1980 and 1990 CSDs. Based on good agreement with literature results in this preliminary modeling, I was comfortable including all these factors as confounding variables in final models of hypothesized geographic SEHSs, presented in Chapter 5. A list of family factors and how they are coded in the final logistic regression modeled is presented in Table 12.

Table 10. Percentage of Missing Cases in Each CSD
If Paternal Age Variable is Included:

	1970		1980		1990	
	N	Percent	N	Percent	N	Percent
Included in Analysis	1,686,366	90.2	3,155,693	87.2	3,460,806	83.1
Missing Cases	182,534	9.8	462,288	12.8	702,111	16.9
Total	1,868,900	100	3,617,981	100.0	4,142,917	100.0

Table 11. Logistic Regression Results for Family Factors in All CSDs: Race, Plurality, Birth Order, and Season. Bold indicates $p \leq .05$.

	B	S.E.	Wald	Sig.	Exp(B)	95.0% C.I.for EXP(B)	
						Lower	Upper
1970 CSD (N=1,844,278)							
Race of Child			37.654	**.000**			
White	−.025	.004	37.594	**.000**	.975	.967	.983
Black	−.007	.011	.369	.544	.993	.972	1.015
Livebirth order	−.004	.001	24.140	**.000**	.996	.994	.998
Season	−.007	.003	5.191	**.023**	.993	.988	.999
Constant	.070	.003	581.453	**.000**	1.073		
1980 CSD (N=3,581,498)							
Race of child			77.381	**.000**			
Black	−.026	.003	76.404	**.000**	.974	.968	.980
Other	−.011	.006	2.861	.091	.989	.977	1.002
Plurality			35.147	**.000**			
Twins	−.048	.008	34.582	**.000**	.954	.939	.969
>Twins	−.044	.057	.600	.438	.957	.856	1.070
Live-birth order	−.003	.001	13.739	**.000**	.997	.995	.998
Season	−.003	.002	1.695	.136	.997	.993	1.001
Constant	.064	.002	786.073	**.000**	1.067		
1990 CSD (N=4,136,334)							
Race of child			80.042	**.000**			
Black	−.023	.003	75.903	**.000**	.977	.972	.982
Other	−.005	.005	1.196	.274	1.005	.996	1.014
Plurality			18.423	**.000**			
Twins	−.028	.007	17.638	**.000**	.973	.960	.985
Triplets	−.015	.038	.162	.687	.985	.297	1.061
Quads	.117	.148	.630	.982	1.013	.328	3.131
Live-birth order	−.004	.001	26.871	**.000**	.996	.994	.997
Season	−.001	.002	.840	.359	.998	.994	1.002
Constant	.062	.002	824.685	**.000**	1.186		

Table 12. Coding for Family Factor Variables Selected for Multivariate Logistic Regression.

Variable Name	Coding	Indicator member of category
Plurality	Categorical	1970: N/A (Missing); 1980: Triplet or greater value; 1990: Quadruplets.
Live-birth order	Numerical	N/A
Season of birth	Categorical	Summer-Summer month of birth (March, April, May, June, July, and August)

Chapter 5. Results of Logistic Regression for Socio-environmental Hormonal Stressor Variables

U.S. County Urban-Rural Percentages

Moller (1996), Allan et al. (1997), Davis et al. (1998) and others have hypothesized that a small but significant decline in the male proportion of the human sex ratio at birth in the U.S. and several industrialized nations since 1970 is the result of the pervasive presence of environmental toxins that disrupt the human endocrine system. A number of studies since that time have failed to provide supporting evidence of widespread environmental effects in the general population, although high levels of exposure to dioxins will almost certainly lower the male proportion of births (Mocarelli et al. 1996), and other compounds are also suspected.

Some researchers have examined SRBs in highly agricultural areas to see if presumed higher exposure to agricultural pesticides reduces the SRB (e.g., Garry et al. 2002). Others have found some evidence that the urban environment would be more likely to reduce the SRB (Astolfi and Zonta 1999a). Complicating exploration of their competing effects, if they exist, is the possibility that exposure in the general population to environmental estrogens may be geographically diffuse due to exposure routes via the steroid compounds residing in meat products; residual metabolites generated from pesticides on fruits and vegetables; dioxin compounds leached into milk from paper containers; chemically unstable plastics used as food wraps; and endocrine disrupting contaminants from other common foodstuffs and containers.

Differential rural/urban SRBs have been noted for many decades, but James (1987a) dismissed their significance in his comprehensive review of SRB factors. He did, however, note that the rate of dizygotic twinning, which has been shown to correlate to gonadotropin levels, appears to have increased worldwide in rural areas. From this and other studies cited in the literature review, I hypothesized that a differential urban/rural SRB might be detectable geographically at the U.S. county level. I hypothesized that counties with high farm population percentages represent a higher risk of exposure to agricultural chemicals than do those with low percentages, and therefore the SRB should be significantly lower in these counties due to endocrine disruption. Further, I predicted

that SRB in urban populations would be higher. Tables 13 and 14 show the results of a univariate logistic regression of farm population percentage and urban population percentage for each county. For the 1990 CSD, an urban population percentage value is not present in the city-county data compilation but was available from STF-3 data summaries published separately by the U.S. Census. Table 15 reports logistic regression results for a multivariate model of family factors and urban/farm percentages for white births only in all CSDs. Table 16 reports logistic regression results for black births only for these CSDs. As these results show, the SRB bias is the opposite of my hypothesis for both univariate and multivariate models.

U.S. County Reproductive Adult Sex Ratios (RASRs)

Lummaa et al. (1998) found that the SRB in Finnish church parishes from 1775 to 1850 was negatively correlated to the sex ratio of the adult reproductive population, which they defined as males and females aged 15 to 50. This, they suggested, supported a theory of adaptive response showing that humans facultatively manipulate SRB to give the rarer sex an advantage in the next generation. While adults that define of RASR were not likely partners for the new generation of newborns, the RASR served as the only cue available for hormonal response to adjust the SRB. Some findings correlating adult population sex ratio with births in succeeding years support the operation of such a mechanism (Lazarus 2002). James (2000) examined the populations of 41 countries in decadal intervals over a 50-year span and found no conclusive support for this theory, although some national populations did exhibit this effect. He suggested that study of this effect was more likely to be evident in smaller geographic areas such as U.S. states. If a hormonal mechanism exists, it operates as a parental perception of local population conditions.

Does the SRB adjust in relation to the adult sex ratio so that, for example, more girls are born when they are rare in the adult reproductive age population? If parental perception is critical to hormonal response, a smaller geographic unit is a more suitable observation unit than the nation or U.S. state. While personal perception of the local proportion of reproductive adults may not match the measured proportion, it is more likely to be accurate than for larger proximate areas. To test this hypothesis, I added to

Table 13. Univariate LR Results of County Farm Population Percentage in all CSDs.

	White births			Black births		
	Wald	Sig.	Exp(B)	Wald	Sig.	Exp(B)
1970 CSD						
FarmPopPercent	.901	.342	1.021	1.406	.236	.921
Constant	938.221	.000	1.058	60.517	.000	1.033
1980 CSD						
FarmPopPercent	2.732	.098	1.038	1.271	.260	.887
Constant	1689.430	.000	1.057	106.922	.000	1.031
1990 CSD						
FarmPopPercent	4.254	**.039**	1.001	.129	.720	1.001
Constant	1698.516	.000	1.053	116.570	.000	1.029

Table 14. Univariate LR Results of County Urban Population Percentage in all CSDs.

	White births			Black births		
	Wald	Sig.	Exp(B)	Wald	Sig.	Exp(B)
1970 CSD						
UrbanPopPercent	.115	.734	1.000	1.188	.276	1.000
Constant	154.715	.000	1.058	2.741	.098	1.019
1980 CSD						
UrbanPopPercent	5.774	**.016**	.990	.132	.717	.996
Constant	372.014	.000	1.065	12.914	.000	1.032
1990 CSD						
UrbanPopPercent	6.912	**.009**	1.000	.170	.680	1.000
Constant	335.052	.000	1.062	14.047	.000	1.032

Table 15. White Birth LR Multivariate Model of Family Factors and Urban or Farm Population Percentage.

	B	S.E.	Wald	Sig.	Exp(B)	95.0% C.I. for EXP(B)	
						Lower	Upper
1970 (N=1528208)							
Live-birth order	−.004	.001	15.383	**.000**	.996	.994	.998
Season	−.008	.003	5.403	**.020**	.993	.986	.999
FarmPopPercent	.025	.022	1.231	.267	1.025	.981	1.070
Constant	.069	.003	432.165	**.000**	1.071		
1970 (N=1528208)							
Live-birth order	−.004	.001	14.913	**.000**	.996	.994	.998
Season	−.008	.003	5.384	**.020**	.993	.986	.999
UrbanPopPercent	.000	.000	.061	.805	1.000	1.000	1.000
Constant	.069	.005	162.770	**.000**	1.071		
1980 (N=2860976)							
Plurality			26.277	**.000**			
Live-birth order	−.004	.001	19.807	**.000**	.996	.994	.998
Season	−.003	.002	1.846	.174	.997	.992	1.001
FarmPopPercent	.042	.023	3.377	.066	1.043	.997	1.090
Constant	.039	.061	.401	.526	1.039		
1980 (N=2860976)							
Plurality			26.232	**.000**			
Live-birth order	−.004	.001	19.657	**.000**	.996	.994	.998
Season	−.003	.002	1.846	.174	.997	.992	1.001
UrbanPopPercent	−.011	.004	7.498	**.006**	.989	.981	.997
Constant	.048	.061	.628	.428	1.050		
1990 (N=3206841)							
Plurality			17.615	**.001**			
Live-birth order	−.005	.001	24.182	**.000**	.995	.993	.997
Season	.000	.002	.019	.892	1.000	.995	1.004
FarmPopPercent	.001	.000	4.775	**.029**	1.001	1.000	1.001
Constant	.194	.606	.103	.748	1.214		
1990 (N=3206841)							
Plurality			17.635	**.001**			
Live-birth order	−.005	.001	23.564	**.000**	.995	.994	.997
Season	.000	.002	.018	.894	1.000	.995	1.004
UrbanPopPercent	.000	.000	7.042	**.008**	1.000	1.000	1.000
Constant	.205	.606	.114	.736	1.227		

Table 16. Black Birth LR Multivariate Model of Family Factors and Urban or Farm Population Percentage.

	B	S.E.	Wald	Sig.	Exp(B)	95.0% C.I. for EXP(B)	
						Lower	Upper
1970 Farm Population Percent (N=282495)							
Live-birth order	–.004	.002	6.281	**.012**	.996	.993	.999
Season	–.002	.008	.102	.750	.998	.983	1.012
FarmPopPercent	–.059	.070	.703	.402	.943	.821	1.082
Constant	.044	.007	37.979	**.000**	1.046		
1970 Urban Population Percent (N=282495)							
Live-birth order	–.004	.002	6.310	**.012**	.996	.993	.999
Season	–.002	.008	.099	.753	.998	.983	1.012
UrbanPopPercent	.000	.000	.605	.437	1.000	1.000	1.000
Constant	.035	.013	6.852	**.009**	1.035		
1980 Farm Population Percent (N=582960)							
Plurality			18.034	**.000**			
Live-birth order	–.003	.002	2.775	.096	.997	.994	1.001
Season	.000	.005	.002	.964	1.000	.989	1.010
FarmPopPercent	–.085	.107	.629	.428	.918	.744	1.134
Constant	–.053	.139	.148	.700	.948		
1980 Urban Population Percent (N=582960)							
Plurality			17.986	**.000**			
Live-birth order	–.003	.002	3.070	.080	.997	.993	1.000
Season	.000	.005	.003	.958	1.000	.989	1.010
UrbanPopPercent	–.007	.010	.507	.477	.993	.974	1.013
Constant	–.048	.139	.118	.731	.953		
1990 Farm Population Percent (N=717455)							
Plurality			4.719	.194			
Live-birth order	–.004	.002	4.509	**.034**	.996	.993	1.000
Season	–.008	.005	2.520	.112	.992	983	1.002
FarmPopPercent	.001	.002	.122	.726	1.001	997	1.004
Constant	.308	.441	487	.485	1.361		
1990 Urban Population Percent (N= 717455)							
Plurality			4.718	.194			
Live–birth order	–.004	.002	4.501	**.034**	996	993	1.000
Season	–.008	.005	2.524	.112	.992	.983	1.002
UrbanPopPercent	.000	000	.193	.661	1.000	1.000	1.000
Constant	.312	.441	.500	.479	1.366		

the CSD an RASR variable defining reproductive age as persons aged 15 to 49 years old, the upper and lower bound correlating with the five-year age divisions published by U.S. Census county summaries; this is slightly narrower than the Lummaa et al. (1998) definition of reproductive age (15–50), and slightly broader than that adopted by James (2000) of 15–44. The RASR value for each birth record was calculated as the number of males of reproductive age in the county divided by the total males and females of reproductive age in the county.

Table 17 presents the results of univariate logistic regression of the RASR and SRB in all CSDs. Table 18 displays results of an SRB multivariate model using family factors and the RASR value for white births in all CSDs. Table 19 displays results for the same model using black births only. The RASR variable approaches significance for black births in 1980 only, in both univariate and multivariate models. While nonsignificant, the SRB bias in white births is the opposite of that hypothesized: increases in the RASR increase the SRB.

U.S. County White and Black Population Percentages

James (1987b) suggested that societal status may influence gonadotropin and testosterone levels, perhaps explaining the consistently lower SRB of blacks in populations of the U.S. and European nations. The percentages of white and black populations vary significantly in the 3106 counties of my dataset. Given the long and unresolved history of racial strife in the U.S., the strength of racial differentiation in a local environment might lead to a corresponding response in the hormonal environment of members of a racial group. This may be reflected in both white and black SRBs in relation to their relative percentage of total county population. While other racial grouping might also be employed in this analysis, the wide geographic distribution of white and black groupings is available in all sample years and serves as an initial benchmark for the presence of such an influence. If racial minority status does depress testosterone and increase gonadotropin levels, I hypothesized that black SRBs would increase in counties where the percentage of black population approached majority status. As a corollary, I expected that white SRB values would decrease as the proportion of white population in the county decreases. To test this model I added to the CSDs the

Table 17. Univariate LR Results of County Reproductive Adult Sex Ratio in all CSDs.

	White births			Black births		
	Wald	Sig.	Exp(B)	Wald	Sig.	Exp(B)
1970 CSD						
AdultReproRatio	.807	.369	1.067	1.898	.168	1.262
Constant	.530	.467	1.026	1.016	.313	.921
1980 CSD						
AdultReproRatio	2.798	.094	1.133	3.383	.066	.774
Constant	.025	.874	.994	5.095	.024	1.167
1990 CSD						
AdultReproRatio	2.038	.153	1.104	.510	.475	.912
Constant	.006	.937	1.003	1.346	.246	1.077

Table 18. White Birth LR Multivariate Model of Family Factors and Reproductive Adult Sex Ratio.

	B	S.E.	Wald	Sig.	Exp(B)	95.0% C.I. for EXP(B)	
						Lower	Upper
1970 (N=1528208)							
Livebirth order	−.004	.001	14.889	**.000**	.996	.994	.998
Season	−.008	.003	5.387	**.020**	.993	.986	.999
AdultReproRatio	.058	.072	.653	.419	1.060	.920	1.222
Constant	.041	.036	1.334	.248	1.042		
1980 (N=2860976)							
Plurality			26.345	**.000**			
Singleton	.027	.061	.200	.655	1.028	.912	1.158
Twins	−.018	.062	.086	.769	.982	.870	1.108
Livebirth order	−.004	.001	19.322	**.000**	.996	.994	.998
Season	−.003	.002	1.842	.175	.997	.992	1.001
AdultReproRatio	.124	.075	2.718	.099	1.131	.977	1.310
Constant	−.022	.072	.094	.760	.978		
1990 (N=3206841)							
Plurality			17.610	**.001**			
Singleton	-.132	.606	.048	.827	.876	.267	2.871
Twins	-.163	.606	.073	.788	.850	.259	2.784
Triplets	-.113	.607	.035	.852	.893	.272	2.933
Quads	.014	.627	.001	.982	1.015	.297	3.465
Livebirth order	-.005	.001	23.867	**.000**	.995	.994	.997
Season	-.001	.002	.253	.615	.999	.995	1.003
AdultReproRatio	.107	.069	2.389	.122	1.113	.972	1.276
Constant	.141	.607	.054	.816	1.152		

Table 19. Black Birth LR Multivariate Model of Family Factors and Reproductive Adult Sex Ratio.

	B	S.E.	Wald	Sig.	Exp(B)	95.0% C.I. for EXP(B)	
						Lower	Upper
1970 (N=282495)							
Livebirth order	–.004	.002	6.689	**.010**	.996	.992	.999
Season	–.002	.008	.106	.745	.998	.983	1.012
AdultReproRatio	.251	.170	2.192	.139	1.285	.922	1.793
Constant	–.079	.083	.904	.342	.924		
1980 (N=582960							
Plurality			17.985	**.000**			
Singleton	.091	.138	.432	.511	1.095	.835	1.437
Twins	.019	.139	.018	.892	1.019	.775	1.339
Livebirth order	–.003	.002	3.051	.081	.997	.993	1.000
Season	.000	.005	.002	.965	1.000	.989	1.010
AdultReproRatio	–.261	.140	3.500	.061	.770	.586	1.013
Constant	.075	.155	.235	.628	1.078		
1990 (N= 717455)							
Plurality			4.701	.195			
Singleton	-.268	441	.369	.544	765	.322	1.816
Twins	-.282	441	.408	.523	.754	.318	1.791
Triplets	-.480	456	1.109	.292	619	.253	1.512
Live-birth order	-.004	002	4.534	**.033**	996	.993	1.000
Season	-.006	005	1.387	.239	994	.985	1.004
AdultReproRatio	-.088	130	.463	.496	916	.710	1.181
Constant	.351	446	.622	.430	.421		

county white and black population percentage fields available from CCDBs. Table 20 shows the results of univariate logistic regression of white population percentage and black population percentage on white births; Table 21 is the same univariate model for black births. Table 22 presents the results of a SRB logistic regression multivariate logistic regression model of family factors and county racial composition for white births in all CSDs. Table 23 presents the same model for black births. These results show consistent increases in SRB with increases of white population percentage for both white and black births; black birth SRB decreases with increases in black population percentage, as does white SRB in two of the three CSDs.

U.S. County Percentages of Families Below Poverty Line

The NCHS dataset does not include any direct indication of parental socioeconomic status. Education of the father is reported, but the high number of missing records with missing father data has already been discussed as reason to exclude those variables from geographic analysis. Education of the mother is frequently present, but I did find sufficient support in literature review for using its value as a surrogate for the socioeconomic status of a family. A number of county economic measures are available in census city-county data compilations, including per capita and median family income, civilian unemployment rates, and persons or families which fall below defined poverty levels for the year of reporting. Most of these variables correlate highly. All have inherent problems in ranking the socioeconomic status of a county, given the degree to which cost of living varies regionally. Based on results of factor analysis and preliminary regression testing, I selected the county percentage of families whose incomes fall below federally defined standards for poverty as the socioeconomic indicator variable. Based on literature reviewed, I hypothesized that a high level of poverty within a county is a hormonal stressor that will lower the SRB of county residents. Table 24 shows univariate logistic regression results for percentage of families below the poverty line in a county on white and black births. Table 25 presents results of a multivariate model of family factors and the county poverty line percentage for white births; Table 26 provides the results of similar modeling for black births. These results show significant reduction of

Table 20. Univariate LR Results of County White Population Percentage in all CSDs.

	White births			Black births		
	Wald	Sig.	Exp(B)	Wald	Sig.	Exp(B)
1970 CSD						
WhitePopPercent	.469	.225	1.018	.018	.894	1.003
Constant	.633	.002	1.042	2.282	.131	1.028
1980 CSD						
WhitePopPercent	.618	.432	1.007	3.808	**.051**	1.035
Constant	7.193	.000	1.051	.140	.708	1.005
1990 CSD						
WhitePopPercent	.597	**.032**	1.017	.615	.433	1.012
Constant	6.046	.000	1.040	4.182	.041	1.021

Table 21. Univariate LR Results of County Black Population Percentage in all CSDs.

	White births			Black births		
	Wald	Sig.	Exp(B)	Wald	Sig.	Exp(B)
1970 CSD						
BlackPopPercent	.713	.398	.987	.005	.943	.998
Constant	748.468	.000	1.061	20.203	.000	1.031
1980 CSD						
BlackPopPercent	.056	.813	1.003	5.742	**.017**	.961
Constant	1292.465	.000	1.058	63.094	.000	1.039
1990 CSD						
BlackPopPercent	.889	.346	.990	.428	.513	.990
Constant	226.837	.000	1.055	49.981	.000	1.031

Table 22. White Birth LR Multivariate Model of Family Factors and County Racial Composition.

	B	S.E.	Wald	Sig.	Exp(B)	95.0% C.I. for EXP(B)	
						Lower	Upper
1970 (N=1528208)							
Live-birth order	–.004	.001	15.432	**.000**	.996	.994	.998
Season	–.008	.003	5.370	**.020**	.993	.986	.999
WhitePopPercent	.018	.015	1.493	.222	1.018	.989	1.049
Constant	.054	.014	15.441	**.000**	1.055		
1970 (N=1528208)							
Live-birth order	–.004	.001	15.291	**.000**	.996	.994	.998
Season	–.008	.003	5.371	**.020**	.993	.986	.999
BlackPopPercent	–.013	.015	.747	.387	.987	.957	1.017
Constant	.071	.004	399.848	**.000**	1.074		
1980 (N=2860976)							
Plurality			26.370	**.000**			
Live-birth order	–.004	.001	19.294	**.000**	.996	.994	.998
Season	–.003	.002	1.838	.175	.997	.992	1.001
WhitePopPercent	.009	.009	.837	.360	1.009	.990	1.028
Constant	.032	.062	.273	.601	1.033		
1980 (N=2860976)							
Plurality			26.383	**.000**			
Live-birth order	–.004	.001	19.211	**.000**	.996	.994	.998
Season	–.003	.002	1.845	.174	.997	.992	1.001
BlackPopPercent	.000	.011	.002	.968	1.000	.979	1.023
Constant	.040	.061	.420	.517	1.040		
1990 (N=3206841)							
Plurality			16.881	**.002**			
Quads	.014	.627	.000	.982	1.014	.297	3.463
Live-birth order	–.005	.001	22.620	**.000**	.995	.994	.997
Season	.000	.002	.006	.941	1.000	.995	1.004
WhitePopPercent	.017	.008	4.722	**.030**	1.017	1.002	1.033
Constant	.180	.606	.088	.766	1.197		
1990 (N=3206841)							
Plurality			17.664	**.001**			
Live-birth order	–.005	.001	23.829	**.000**	.995	.994	.997
Season	.000	.002	.018	.893	1.000	.995	1.004
BlackPopPercent	–.011	.010	1.153	.283	.989	.969	1.009
Constant	.195	.606	.104	.747	1.216		

Table 23. Black Birth LR Multivariate Model of Family Factors and County Racial Composition.

	B	S.E.	Wald	Sig.	Exp(B)	95.0% C.I. for EXP(B)	
						Lower	Upper
1970 (N=282495)							
Live-birth order	–.004	.002	6.690	**.010**	.996	.992	.999
Season	–.002	.008	.104	.747	.998	.983	1.012
WhitePopPercent	.003	.024	.018	.893	1.003	.957	1.052
Constant	.041	.020	4.324	.038	1.042		
1970 (N=282495)							
Live-birth order	–.004	.002	6.703	**.010**	.996	.992	.999
Season	–.002	.008	.104	.747	.998	.983	1.012
BlackPopPercent	–.001	.024	.003	.954	.999	.953	1.047
Constant	.044	.009	23.628	.000	1.045		
1980 (N=582960)							
Plurality			18.042	**.000**			
Live-birth order	–.003	.002	2.785	.095	.997	.994	1.001
Season	.000	.005	.002	.960	1.000	.989	1.010
WhitePopPercent	.033	.018	3.540	.060	1.034	.999	1.070
Constant	–.078	.139	.313	.576	.925		
1980 (N=582960)							
Plurality			18.036	**.000**			
Live-birth order	–.003	.002	2.639	.104	.997	.994	1.001
Season	.000	.005	.002	.961	1.000	.989	1.010
BlackPopPercent	–.038	.017	5.174	**.023**	.962	.931	.995
Constant	–.046	.139	.108	.743	.955		
1990 (N= 717455)							
Plurality			4.727	.193			
Live-birth order	–.004	.002	4.484	**.034**	.996	.993	1.000
Season	–.008	.005	2.512	.113	.992	.983	1.002
WhitePopPercent	.013	.015	.779	.377	1.013	.984	1.043
Constant	.301	.441	.466	.495	1.351		
1990 (N= 717455)							
Plurality			4.732	.193			
Live-birth order	–.004	.002	4.476	**.034**	.996	.993	1.000
Season	–.008	.005	2.506	.113	.992	.983	1.002
BlackPopPercent	–.010	.015	.496	.481	.990	.962	1.019
Constant	.312	.441	.501	.479	1.366		

Table 24. Univariate LR Results of County Families Below Poverty Line Percentage in all CSDs.

	White births			Black births		
	Wald	Sig.	Exp(B)	Wald	Sig.	Exp(B)
1970 CSD						
BelowPOVLine	4.446	**.035**	.999	.887	.346	1.000
Constant	436.454	.000	1.065	26.497	.000	1.036
1980 CSD						
BelowPOVLine	.987	.321	.949	2.428	.119	.881
Constant	598.448	.000	1.060	40.033	.000	1.038
1990 CSD						
BelowPOVLine	4.512	**.034**	1.000	1.012	.315	1.000
Constant	611.046	.000	1.058	38.125	.000	1.034

Table 25. White Birth LR Multivariate Model of Family Factors and County Percentage Below Poverty Line.

	B	S.E.	Wald	Sig.	Exp(B)	95.0% C.I. for EXP(B)	
						Lower	Upper
1970 (N=1528208)							
Live-birth order	−.004	.001	14.623	**.000**	.996	.994	.998
Season	−.007	.003	5.281	**.022**	.993	.986	.999
PerBelowPOV	.000	.000	3.828	**.050**	1.000	.999	1.000
Constant	.075	.004	338.700	**.000**	1.078		
1980 (N=2860976)							
Plurality			26.360	**.000**			
Singleton	.027	.061	.200	.655	1.028	.912	1.158
Twins	−.018	.062	.086	.769	.982	.870	1.108
Live-birth order	−.004	.001	19.255	**.000**	.996	.994	.998
Season	−.003	.002	1.841	.175	.997	.992	1.001
PerBelowPOV	−.056	.053	1.127	.288	.945	.852	1.049
Constant	.042	.061	.470	.493	1.043		
1990 (N=3206841)							
Plurality			17.823	**.001**			
Singleton	−.131	.606	.047	.829	.877	.268	2.875
Twins	−.162	.606	.072	.789	.850	.260	2.787
Triplets	−.113	.607	.035	.852	.893	.272	2.935
Quads	.014	.627	.001	.982	1.015	.297	3.465
Live-birth order	−.005	.001	22.748	**.000**	.995	.994	.997
Season	.000	.002	.015	.904	1.000	.995	1.004
PerBelowPOV	.000	.000	4.167	**.041**	1.000	.999	1.000
Constant	.197	.606	.106	.745	1.218		

Table 26. White Birth LR Multivariate Model of Family Factors and County Percentage Below Poverty Line.

	B	S.E.	Wald	Sig.	Exp(B)	95.0% C.I. for EXP(B)	
						Lower	Upper
1970 (N=282495)							
Live-birth order	–.004	.002	6.278	**.012**	.996	.993	.999
Season	–.002	.008	.100	.752	.998	.983	1.012
PerBelowPOV	.000	.000	.448	.503	1.000	.999	1.001
Constant	.047	.009	27.874	**.000**	1.048		
1980 (N=582960)							
Plurality			18.052	**.000**			
Singleton	.092	.138	.440	.507	1.096	.836	1.438
Twins	.020	.139	.020	.888	1.020	.776	1.340
Live-birth order	–.003	.002	2.782	.095	.997	.994	1.001
Season	.000	.005	.002	.961	1.000	.989	1.010
PerBelowPOV	–.124	.082	2.317	.128	.883	.752	1.036
Constant	–.047	.139	.113	.737	.954		
1990 (N= 717455)							
Plurality			4.740	.192			
Singleton	–.269	.441	.372	.542	.764	.322	1.814
Twins	–.283	.441	.413	.521	.753	.317	1.788
Triplets	–.481	.456	1.114	.291	.618	.253	1.510
Live-birth order	–.004	.002	4.319	**.038**	.996	.993	1.000
Season	–.008	.005	2.498	.114	.992	.983	1.002
PerBelowPOV	.000	.000	1.183	.277	1.000	.999	1.000
Constant	.314	.441	.508	.476	1.370		

SRB in white births as this hormonal stressor increases; however, this effect is not present or not significant for black births.

Combined model with all SEHS variables

As a partial measure of the relationship among the evaluated SEHS variables, I applied stepwise logistic regression methods on multivariate models that included family factor variables and each of the SEHS variables analyzed in separate models presented above. These include plurality (except for the 1970 CSD), livebirth order, season of birth, farm population percentage, urban population percentage, percent of population below poverty level, RASR, percentage of black population, and percentage of white population (plurality is missing from the 1970 dataset). In addition, I ran all these models separately with the addition of U.S. regional division (see Table 1) as a variable to evaluate whether regional differences noted as significant in previous studies (Marcus et. al. 1998; Allen et. al 1997) remain significant when the demographic factors I have considered are present.

To reduce the model to the fewest terms I applied forward stepwise regression with entry testing based on the significance of the score statistic, and removal testing based on the probability of a likelihood-ratio statistic based on conditional parameter estimates (SPSS 2004). I also applied backwards stepwise selection, in which removal testing is based on the probability of the likelihood-ratio statistic based on conditional parameter estimates (SPSS 2004); this method was used to detect suppressor effects. Where the forward and backwards stepwise methods produce the same model terms, and where the addition of the regional division does not change model results, I adopted the forward stepwise regression results as the fitted model for the CSD. Where these results differ, I adopted the model based on other selection criteria, as described below for each model.

1970 White SEHS Model

The 1970 White CSD SEHS model has 1,528,208 observations, 98.9 percent of all white births recorded in 1970 by NCHS with the CSD geographic area (excluding Alaska and residents of countries outside the U.S). Forward stepwise regression preserved only live-birth order and season of birth as significant variables; backward

stepwise regression added percentage of population below poverty level and farm population percentage to these variables. The addition of the U.S. regional division did not affect these results. A model with season of birth removed was also run, again returning percentage of population below poverty level and farm population percentage as significant, along with live-birth order. However, the first model was selected because it had the fewest variables and the lowest –2LL value.

1970 Black SEHS Model

The 1970 Black CSD SEHS model has 282,495 observations, 98.7 percent of all black birth cases reported by NCHS with the CSD geographic area. In all model runs, live-birth order is the only significant variable.

1980 White SEHS Model

The 1980 White CSD SEHS model has 2,892,112 observations, 99.8 percent of all white birth cases reported by NCHS. All model procedures reduced the variable set to plurality, live-birth order, and urban population percentage.

1980 Black SEHS Model

The 1980 black birth combined variable dataset has 582,960 observations, 98.9 percent of all black birth cases within the CSD geographic area. The forward selection method preserved plurality and percentage of black population as significant variables. The backwards regression procedure added farm population percentage, urban population percentage and RASR as significant variables. Addition of the U.S. region did not change these results. The forward selection model was selected because of its efficiency, although it had a slightly higher –2LL than the backwards selection model.

1990 White SEHS Model

The 1990 White CSD SEHS model has 3,206,750 observations, 99.3 percent of all white births recorded in 1990 by NCHS with the CSD geographic area. The forward selection and backward selection methods both selected percentage of population below poverty line and urban population percentage as significant variables, along with plurality and live-birth order. Addition of U.S. division, however, changed the results of the forward selection method to RASR and U.S. region, with plurality and live-birth order. The backwards selection model with U.S. region included, however, retained division,

but replaced RASR with percentage below poverty line and percent urban population as significant. Therefore, the forward selection model was selected because of suspected suppressor effects elevating RASR to significance, although the –2LL value was slightly higher for the forward selection model without U.S. region included.

1990 Black SEHS Model

The 1990 White CSD SEHS model has 717,389 observations, 99 percent of all black births recorded in 1990 by NCHS with the CSD geographic area. All model procedures preserved only the live-birth order variable.

Results for white birth CSDs are reported in Table 27; for black birth CSDs, in Table 28. A discussion of these results and the conclusion of my thesis follow in Chapter 6.

Table 27. White Birth Forward Stepwise LR Multivariate Model of All Hypothesized SEHS and Family Factors.

	B	S.E.	Wald	Sig.	Exp(B)	95.0% C.I. for EXP(B)	
						Lower	Upper
1970 (N = 1,528,208)							
Live-birth order	–.004	.001	14.879	**.000**	.996	.994	.998
Season	–.008	.003	6.471	**.011**	.992	.985	.998
Constant	.070	.003	453.529	**.000**	1.073		
1980 (N=2,892,112)							
Plurality			22.285	**.000**			
Singleton	.005	.063	.007	.933	.005	.889	1.137
Twins	-.038	.064	.363	.547	.962	.850	1.090
Live-birth order	-.004	.001	15.630	**.000**	.996	.994	.998
UrbanPopPercent	-.011	.004	6.142	**.013**	.989	.981	.998
Constant	.067	.063	1.135	.287	.069		
1990 (N=3,206,750)							
Plurality			17.757	**.000**			
Singleton	-.146	.161	.814	.367	.865	.630	1.186
Twins	-.177	.162	1.196	.274	.838	.611	1.150
Triplets	-.127	.166	.585	.444	.880	.635	1.220
Live-birth order	-.005	.001	22.655	**.000**	.995	.994	.997
PerBelowPov	-.001	.000	6.179	**.013**	.999	.999	1.000
PercentUrban	.000	.000	9.071	**.003**	1.000	1.000	1.000
Constant	.222	.161	1.896	.169	.249		

Table 28. Black Birth Forward Stepwise LR Multivariate Model of All Hypothesized SEHS and Family Factors.

	B	S.E.	Wald	Sig.	Exp(B)	95.0% C.I. for EXP(B)	
						Lower	Upper
1970 (N=282,495)							
Live-birth order	-.004	.002	6.737	**.009**	.996	.992	.999
Constant	.042	.006	50.590	.000	1.043		
1980 (N=582,960)							
Plurality			13.959	**.001**			
Singleton	.093	.144	.416	.519	1.097	.828	1.454
Twins	.027	.145	.035	.851	1.028	.774	1.365
BlackPopPercent	-.038	.018	4.547	**.033**	.962	.929	.997
Constant	-.053	.144	.136	.712	.948		
1990 (N=717,389)							
Live-birth order	-.004	.002	5.028	**.025**	.996	993	1.000
Constant	.037	.004	69.354	.000	1.038		

Chapter 6. Discussion and Conclusions

As the results presented in the previous chapter suggest, there are indicators of significant influences on the human sex ratio at birth (SRB) from my hypothesized socio-environmental hormonal stressors (SEHSs). In this final chapter I will summarize the findings of modeling the SEHS variables and assess their individual influences on the SRB, along with their relationships to other SEHS variables modeled. I will assess these findings in light of my underlying hypothesis of significant external hormonal stressors that may find expression at local geographic scales, and I will suggest further experiments that might lend more clarity to the nature of influences whose outlines are indicated only vaguely by the results presented herein.

Urban and Rural SRB Influences

As summarized in Table 29, my multivariate logistic regression models showed that white SRB is negatively associated with the urban population proportion of the county of residence, and positively associated with the farm population proportion. In multivariate models, white SRB slightly but significantly increased with higher farm population proportion in the1990 county SRB dataset (CSD) (Odds Ratio (OR) =1.001; p=0.029) and nearly significantly in 1980 (OR=1.043; p=.098). A nonsignificant (p=0.267) white SRB increase associated with this variable was also found in 1970 (OR=1.025). Influences of urban population proportion on white SRB trailed those of farm population proportion. Its influence was significant in 1990 (p=.008) and 1980 (p=.006), but with nearly neutral ORs in all three years (1990: 1.000; 1980: .998; and 1970: 1.000). Based on the confidence intervals for these values, it appears that there was a very slight decrease in OR associated with urban population proportion—less than .001.

I expected that black SRB would respond in parallel to white SRB in farm versus urban environments, based on the oversimplified assumption that both racial groups are exposed similarly to any stressors present in these distinct environments. However, no significant influences on black SRB were found related to either urban or farm population proportions. Whereas farm population proportion appeared to increase white SRB in all

Table 29. Summary of LR Multivariate Model of Family Factors and County Farm/Urban Population Percentage in all CSDs

	White			Black		
	Wald	Sig.	Odds Ratio	Wald	Sig.	Odds Ratio
Farm Population						
1970	1.231	.267	1.025	.703	.402	.943
1980	3.377	.066	1.043	.629	.428	.918
1990	4.775	**.029**	1.001	.041	.840	1.000
Urban Population						
1970	.061	.805	1.000	.605	.437	1.000
1980	7.498	**.006**	998	.507	.477	.993
1990	7.042	**.008**	1.000	.069	.793	1.000

three CSDs, it appeared to decrease black SRB in 1970 and 1980, with a slight increase in 1990. Urban population percentage was neutral as an influence on black SRB in 1970 and 1990, and appeared to decrease black SRB in 1980 (OR=.996); these results are closer to the white SRB results than the effect of farm population proportion.

Modeled as a single variable, farm population was only significant (OR=1.001; p=.029) in 1990 for white SRB. The univariate model of urban population and white SRB was significant in 1980 (p=.009) and 1990 (p=.016). Neither farm population nor urban population was significant in univariate models for black births in any CSD.

A positive association between white SRB and county farm population proportion runs counter to my hypothesis that births in such counties might be more influenced by exposure to agricultural chemicals and, thus, hormonal disruptions that might lower the male proportion of births. However, my finding is similar to the decline found in major Italian cities by Astolfi and Zonta (1999a) during the period 1970 to 1995. These authors correlated metropolitan areas with dense industrialization and the possibility of increased exposure to environmental toxins. Their data confirmed a hypothesis that, while medical care had been better in metropolitan areas, such areas had undergone "environmental deterioration typical of highly developed countries." They also noted that stillbirth incidence had risen in urban areas, another indicator suggesting that environmental conditions in such areas might be stressing males disproportionately. A related possible cause of lower SRB in urban areas, they speculated, was that the more highly educated and employed women in urban areas were likely to marry and bear children later in life, thus invoking the parental age effect. My multivariate model controls for birth order, so I am inclined to discount this demographic explanation as the cause for what appears to be a very slightly lower SRB in urban areas compared to areas of farm population in the three CSDs I evaluated.

Without further evidence, however, it is difficult to attribute this slight urban disadvantage in SRB to an environment "somehow impaired," as Astolfi and Zonta describe the dense and industrialized urban setting. A slight increase in SRB among rural blacks and whites compared to their urban counterparts was found as early as 1931 by Sanford Winston. Differing environmental toxin exposures are only one of several

conditions of urban living that might differ from other areas; RASR, socioeconomic conditions, and racial composition are three that I have also examined. Urban black populations would likely be more exposed to industrial toxins than would urban whites, based on several investigations of racial minority disadvantage in industrial siting and pollution control decisions, collectively termed as environmental justice. However, while the SRB of whites decreases slightly in the urban environment, the SRB of blacks does not appear to be as significantly affected; black SRB does not rise, at any rate, as farm population increases. Black SRB appears to be subject to different influences than white SRB, or is differently influenced by the same factors that affect white SRB.

RASR

Another factor that might differ between urban and farm environments is the reproductive adult sex ratio, or RASR. In my CSDs, the percentage of the total U.S. population within the age range I used to calculate RASR (15–49) was 47 percent of the total population in 1970, 50.6 percent in 1980, and 52.8 percent in 1990. The effect of RASR on SRB in my results is not as convincing as that of the urban/farm environment difference, but some interesting patterns may be seen in the summary of results in Table 30. RASR is not significant as a univariate influence on SRB for either black or white births, but is significant in a multivariate model for whites in 1990 (OR=1.043, p=.043). This positive association between white SRB and RASR is counter to the findings of Lummaa et al. (1998), who saw decreases in SRB as RASR increased in historical Finnish church parishes. White SRB also increased in parallel with RASR in 1970 (OR=1.060, p=.419) and 1980 (OR=1.131; p=.099), although not significantly. The less-than-one OR of black SRB in 1980 (OR=.770; p=.061) and 1990 (OR=.916, p=.496) is closer to the predicted SRB bias, but is not significant in any of my models.

These results do not confirm my hypothesis that SRB would be negatively correlated to county RASR as mothers facultatively manipulate the sex of their children to the rarer adult sex. There may be several reasons for this. In testing my RASR hypothesis I did not examine other age cohorts as candidate optimal RASR-defining populations. The 15–44 age range, for example, was used by James (2000) in his RASR

Table 30. Summary of LR Multivariate Model of Family Factors and County RASR in all CSDs.

	White			Black		
	Wald	Sign.	Odds Ratio	Wald	Sign.	Odds Ratio
1970	.653	.419	1.060	2.192	.139	1.285
1980	2.718	.099	1.131	3.500	.061	.770
1990	2.389	.122	1.113	.463	.496	.916

study and is also considered the principal age group defining the fertile population by NCHS in its birth statistics tabulations. Also, my calculation of RASR aggregates all racial groups—this total population RASR served as an independent variable for separate white and black birth models. It is possible that racially specific RASR values (i.e., white male to white female percentage) would yield different results. However, my preliminary testing does not support this—white SRB also appears to be positively associated with white RASR. On the other hand, total population RASR does appear to be somewhat correlated with the black population proportion of the county. As Figure 11 shows, the lowest total RASR values are in the South, and closely follow the distribution of black population percentage. The most likely explanation for this is that low SRB in a county population will be carried through to the adult population, depending on migration factors. High RASR could be therefore be a *dependent* variable in the association with SRB; more men are present in a county's reproductive population simply because more males are born there. If this is true, then RASR-SRB correlation may signal a geographic predilection for male or female SRB bias carried through a generation or more.

Further study of the RASR and SRB could examine the association of these variables at larger geographic scales. Perhaps a homeostatic mechanism is not detectable at the county level, where other sex allocation mechanisms may be operating. A local population with a high RASR would presumably have a higher testosterone index than average, as males compete more aggressively for rarer females. Homeostatic mechanisms may operate across larger populations to achieve parity. In the 1990 CSD, the mean county value of RASR was 0.5004, very near the parity predicted by Fisherian sex allocation theory. A casual observation of county SRB mapping shows that opposite extremes of county SRB values are often closely proximate, perhaps indicating an intracounty homeostatic adjustment to nearby imbalances. Spatial autocorrelation of county SRB with weighting for RASR and other confounding factors might reveal more about this relationship.

The changing value of the RASR may also be significant. The RASR of the total U.S. population was .490 in 1970, .496 in 1980, and .505 in 1990. This trend was also noted by Ulizzi and Zonta (1993, 2001) in the U.S., as well as Italy. They speculated that

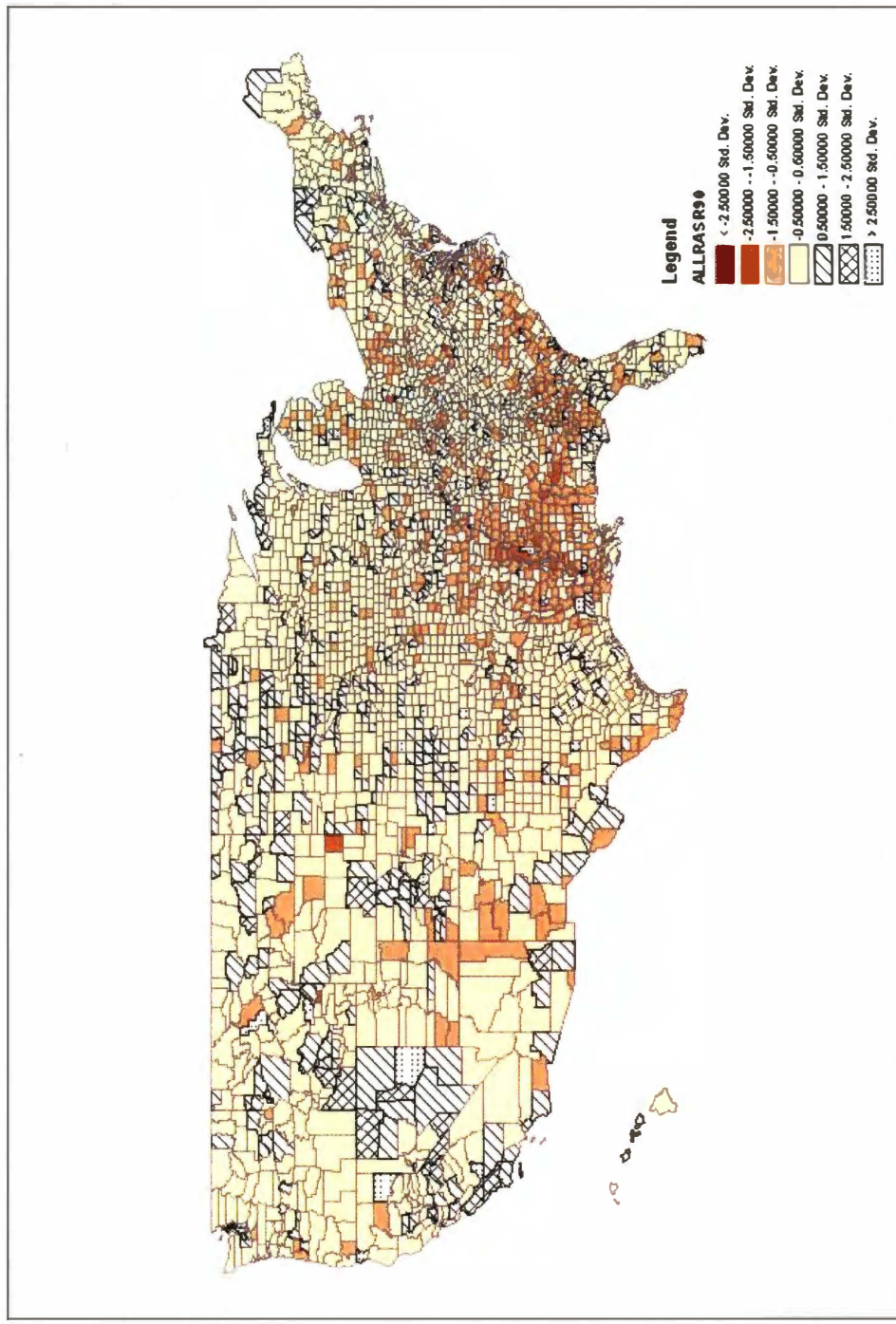


Figure 11. 1990 reproductive adult sex ratio (age 15-49)

reduction in the SRB should be observed as a result of improved survival of males based on Fisherian principles. If the RASR is significantly biased toward males, SRB should adjust to produce fewer of them, so the increase in RASR since 1970 may be an explanation for the decline in the U.S. SRB.

Regional variations in migration and age distribution would result in different RASR dynamics, however. During the study period, for example, the Northeast and the Midwest experienced significant outmigration relative to the South and West, although the Northeast experienced less net outmigration than the Midwest due to a large inmigration from outside the U.S (U.S. Census 1991). Metropolitan areas also experienced a net in-migration compared to nonmetropolitan areas in the 1980s and 1990s, after experiencing net outmigration in the 1970s (U.S. Census 1991). Age, race, and sex characteristics of regional mobility should be considered in further study of RASR-SRB. I observed in Chapter 1 that the decline in U.S. SRB appeared not to have been as pronounced in the South as elsewhere, and that the decline in western states was somewhat more notable. It is worth noting that RASR of these regions differs substantially, partly due to the racial factors I noted above and partly due to the fact that the western U.S. has typically had a higher sex ratio than the eastern U.S. due to male in-migration from the eastern U.S. and elsewhere. In the 1990 CSD, the states with the highest RASR are primarily in the western U.S., while those with the lowest RASR are mainly in the South (Table 31). I also note that the 1990 RASR is higher than average in the heavily agricultural regions of the Midwest, suggesting an association with farm population percentage (Figure 11).

Racial composition

The percentage of black population within a county is a significant coefficient in the SRB multivariate model for blacks in 1980 ($p = .023$) but not in any other sample year for either white or black births (Table 32). While only one CSD has a significant result for this variable, the similar direction of the OR in all CSDs is noteworthy—a reduction of SRB in all models (except for 1980, which is neutral). On the other hand, SRB rises in almost all CSDs as white population increases in a county, significantly so in 1990 for white births ($p=.030$) and nearly significantly in 1980 for black births

Table 31. Lowest and Highest State RASRs in 1990.

Lowest values		Highest Values	
State	**RASR**	**State**	**RASR**
Mississippi	0.4893	Alaska	0.5331
Louisiana	0.4907	Hawaii	0.5235
Alabama	0.4922	California	0.5183
Maryland	0.4937	Nevada	0.5182
Ohio	0.4938	North Dakota	0.5154
West Virginia	0.4940	Wyoming	0.5092
Tennessee	0.4943	Arizona	0.5081
Arkansas	0.4945	Kansas	0.5079
Delaware	0.4945	South Dakota	0.5074
New York	0.4948	Washington	0.5060

Table 32. Summary of LR Multivariate Model of Family Factors and County White/Black Population Percentage in all CSDs.

	White			Black		
	Wald	Sign.	Odds Ratio	Wald	Sign.	Odds Ratio
White Population Percentage						
1970	1.493	.222	1.018	.018	.893	1.003
1980	.837	.360	1.009	3.540	.060	1.034
1990	4.722	**.030**	1.017	.779	.377	1.013
Black Population Percentage						
1970	.747	.387	.987	.003	.954	.999
1980	.002	.968	1.000	5.174	**.023**	.962
1990	1.153	.283	.989	.496	.481	.990

(p=.060). White population percentage is also significant as a univariate logistic regression in 1990 for whites (p=.032) and nearly so for blacks in 1980 (p=.051). Univariate regressions of black population percentage were only significant in 1980 for blacks (p=.017), in parallel with the multivariate results.

I had hypothesized that white and black SRB might increase in relation to majority percentages of their respective racial group. However, it appears that a factor collinear with the percentage of white population increases SRB for either racial group, while a factor collinear with the percentage of black population decreases SRB, also for either racial group. One such factor may be the RASR, which as I have shown appears to decrease as the black population percentage of a county increases.

Another factor correlated with race and ethnicity is economic welfare. In general, members of racial and ethnic minorities have lower incomes, live in worse housing, are less apt to be employed and have less education than white populations (U.S. Census 2001). It is possible that the black population percentage of the county is also associated with the general socioeconomic status of the population, signaling lower health care availability for all residents. The idea that economic disadvantage and correspondingly poor access to health care will disadvantage male births begins at least with Sanford Winston (1931). During the 1970 to 1995 period, there have been significant changes in fetal and infant health measures for both white and blacks. Infant mortality (defined as number of deaths of children 1 year and younger per 1,000 births) declined from 20 to 7.6 between 1970 and 1995; the mortality rate for the perinatal period (from 28th week of pregnancy to 28 days after birth) declined from 23 to 7.6 per 1,000 births during this period (Hovert et al. 2001). Mortality rates improved for all races, but there is still excessive mortality for black births compared to white births in all measures. Perinatal mortality for white births declined from 21 to 6.5 per 1,000 from 1970 to 1995; for black births, from 34 to 13.8 per 1,000 during the same period (Hovert et al. 2001). Perhaps these trend differences contribute to an explanation for the increase in black SRB and the decline in white SRB measured by Marcus et al. (1998) during this period. As more newborn males in a population survive, SRB should increase in the short term, as it has for black births. At some point, however, Fisherian mechanisms would be expected to

correct for the increased numbers of males by reducing SRB. It is possible that white and black populations in the U.S. are on different waypoints of such a trajectory, if it exists.

Percent of families below poverty line

County percentage of families below the poverty line is a significant variable for influence on white SRB in 1990 (.041) and 1970 (.050). In both these sample years, however, for both black and white births, the OR is neutral (Table 33). In 1980 this coefficient reduces the male proportion of births nonsignificantly for both races. Modeled univariately, the results are similar: significant increases for white SRB in 1990 (p=.034) and 1970 (p=.035). I judge that this variable has a barely detectable decreasing effect on SRB—although the OR is neutral in 1970 and 1990, the confidence interval is between .999 and 1.000. This apparent influence is similar on both black and white SRB.

This result parallels some other geographic measures of health care. Some studies, for example, have found an association between the incidence of low birthweight and geographic areas of socioeconomic disadvantage (Ounsted 1982, Starfield and Budetti 1985). The association of SRB and socioeconomic status has not been found in some studies (Erickson 1976), but has been detected in others (Teitelbaum 1970, Teitelbaum and Mantel 1971). Catalano (2003) cited the Trivers Willard hypothesis in his finding that severe socioeconomic decline in East Germany in 1991 had resulted in the lowest SRB in over 40 years. My results suggest that the presence of less extreme socioeconomic factors may also depress SRB. However, there remains the possibility that racial composition is a confounding factor in this influence. In 1970, the percentage of U.S. white families living below the poverty line was 8.1; the percentage of U.S. black families in this category was 32.2. This relationship had not changed significantly by 1990, when 9 percent of white families and 31 percent of black families lived below the poverty line. As the black population of a county increases, so likely on average will the percentage of families living below the poverty line, perhaps partially explaining the correlation between increases in black population proportion and low SRB.

Table 33. Summary of LR Multivariate Model of Family Factors and County Families Below Poverty Line Percentage

	White			Black		
	Wald	Sig.	Odds Ratio	Wald	Sig.	Odds Ratio
1970	3.828	**.050**	1.000	.448	.503	1.000
1980	1.127	.288	945	2.317	.128	.883
1990	4.167	**.041**	1.000	1.183	.277	1.000

Combined models

Clearly, some method is needed to determine the independent contribution of hypothesized SEHS variables, since all appear to have some degree of relationship with each other. I found that the model combining all factors provided some insight into these relationships, although many questions remain. Stepwise modeling of all hypothesized SEHSs found that white SRB was reduced by the family poverty factor in 1990, and urban population percentage in 1980; urban population percentage is also significant in 1990, but the OR is neutral. In 1970, no SEHS value is significant, but season of birth is, the only one of the three CSDs in which this variable appears as a significant influence on SRB. As I noted in Chapter 5, the 1970 model also identifies the family poverty factor (as an SRB reducing factor) and farm population percentage (as an SRB increasing factor) as significant variables if season of birth is removed from the 1970 combined SEHS model. In combined models for black SRB, only black population percentage is a significant SEHS, in the 1980 CSD.

These results confirm the SRB-reducing effects in urban environments, which seem to present the most efficient explanation for the contribution of related SEHS factors studied. The family poverty percentage variable is the most consistent predictor of SRB effect in all CSDs. The slight negative bias in SRB from urban environments and socioeconomic condition is supported by some previous investigators. Somewhat parallel influences detected, of racial composition and RASR factors, could also be attributed to socioeconomic stresses. However, it also appears that urban versus farm population environments have some influence on SRB that is independent of race and the family poverty percentage factor, and may represent some element of the RASR and racial composition factors that I have considered but not scaled correctly.

While these results do not support a theory of environmental toxin exposure, they do suggest that further study of the distinction between urban and rural environments might reveal more evidence of contrasting types or levels of hormonal stressors. One route for such study might be a parallel examination of twinning rates, which James (1996b) speculates are also a reflection of varying parental internal hormone

environments. Based on my results for geographic variation of SRB, I would expect, for example, to see lower twinning rates as farm population increased, and higher twinning rates as urban population increased, which would provide further support of a theory of discrete hormonal conditions in these two living settings. Related health measures that might be useful to local geographic study of reproductive hazard include cryptorchidism, testicular cancer, hypospadias, and ectopic pregnancy.

Confirming the consensus of previous studies, my results showed that the most significant SRB influence is race. My results showed that black and white births responded differently to the SEHSs I hypothesized as influences on SRB, as well as to family factors of parental order, plurality and birth order. It is difficult to derive from my results whether this is due to innate genetic differences or other influences that differentially impact black SRB, such as a greater rate of perinatal mortality due to lower socioeconomic status.

The next most significant factor was plurality, with singleton births having an SRB clearly higher than those of twins. Season of birth also proved a significant factor in 1970, but not so in 1980 and 1990. This may be as Seiver (1985) suggested in his study of the seasonality of the volume of births—the comforts of air conditioning and other buffers to temperature and light have combined with our increasingly nature-estranged living experience to mute our sensitivity to these influences. A growing body of evidence identifies a photoperiod sensitivity mechanism seated in the pineal gland and linked to triggers of reproductive controls by hypothalamus and pituitary hormones; rainfall and temperature changes may also be associated with gonadotropin levels. It is likely that my simple model of seasonality is not sufficient to recognize a distinct signal among the other possibly related effects I have examined, such as regional differences, and farm vs. urban environments. Based on some studies, environmental influences on SRB may be better linked to the time of conception, when hormonal controls would have greater influence, rather than to time of birth. I noted that even in my simple model, there was a higher SRB in the spring–summer season in each of the CSDs. Study of rainfall patterns, deviation from mean temperature, and photoperiod changes and other climate-

related effects warrant detailed study of their association with both the volume of births and SRB.

The relative contribution of my hypothesized SEHSs to SRB is difficult to assess from the available information. Like other SRB factors, these factors would be expected to influence the SRB only slightly, as homeostatic mechanisms would correct any large SRB bias. In the 1990 model year, U.S. region of residence was a significant factor, with the southern U.S. showing a significant increase in SRB compared to other regions. Based on the Wald statistic, where parents lived in the U.S. in 1990 was a more significant influence on SRB than live birth order or parental age, and nearly as important as race and plurality. Regional distinctions have also been noted by Marcus et al. (1998) and are apparent in the review of geographic characteristics I presented in Chapter 1.

Of the SEHS factors I proposed that might define these regional differences, none appear to have the individual significance or influence on SRB that family factors do. Socioeconomic status and urban population percentage both appear to depress SRB by less than .001 in white births; socioeconomic status also depresses black SRB by a slight amount; in both cases the Wald statistic for these factors in any CSD is much less than that for regional division differences found in the 1990 CSD. A similarly small increase in SRB can be found in counties as farm population percentage increases. Both white and black SRB increase with white population percentage and decrease with black population percentage, although significantly so only for 1980 blacks. The positive association of white SRB and county RASR may also be associated with the relationship between black and white population percentages, which may in turn be an indicator of lower prospects for adequate local prenatal care and the survivability of the male fetus. Control for individual economic status would help to clarify these relationships. Birthweight is available as an NCHS data field; the association of low birthweight and lower socioeconomic status would make this variable a useful control in future geographic studies of SRB.

In Chapters 2 and 3 I have reviewed the challenges facing investigators who seek convincing evidence for the hypothesis of facultative, adaptive control of human SRB. Hormonal mechanisms have not been worked out in support of such a theory and it is

clear that these mechanisms, if they exist, operate in modern life in concert with facultative nonhormonal manipulation of the SRB. SRB biases may also be attributed to adaptive strategies or nonadaptive ones. The Trivers Willard hypothesis provides a tempting explanation for some variations in human SRB and is one of the organizing principles for recent SRB research but is, in the phrase of Catalano (2003), "empirically controversial." A full discussion of the problems associated with Trivers Willard and human SRB is beyond the scope of this thesis, but a clear summary of the issue is presented by Lazarus (2002). My results do not support the argument made by Lummaa et al. (1998) regarding RASR, but do not necessarily discount such an explanation for the SRB biases detected. I believe study of RASR-SRB relationships remains a useful subject for further geographic study of SRB. However, the study of SRB seasonality and climate/weather influences might provide the most unambiguous evidence of sex allocation mechanisms in humans.

Even though the shape of external hormonal influences on SRB remains indistinct as my study concludes, I hope that this thesis provides support for SRB research at a local geographic scale. Some of the inconsistencies among my CSD results are partly attributable, I believe, to the gradually improving completeness and accuracy of the NCHS dataset since its inception in the late 1960s. Combined with geographic precision in other census data, these recent natality datasets provide a better opportunity to detect the small perturbations in SRB that are likely to compose a sensitive model. The construction of such a model would be, as I proposed in Chapter 1, a salient contribution to human health studies and is in the tradition of the cholera mapping of Max von Pettenkoffer, John Snow and other pioneers of medical geography. In the context of geographic study, SRB is an important element of the spatial and temporal analysis of demographic, mortality and mobility studies that help identify the changing nature of human health ecologies in both developed and developing nations. I also believe that SRB research will play a part in the recently occurring synergy between physical and human geography, which considers the complex interactions of climate change, local weather patterns, disease vectors, environmental toxins, living and working patterns, socioeconomic conditions, and the biological rhythms of human endocrine system. The

problem of the human sex ratio is indeed as intricate as Darwin suggested. Who better to study it than a geographer?

REFERENCES

REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR). 2003. *CERCLA Priority List of Hazardous Substances for 2003.* Centers for Disease Control. Division of Toxicology. Available at: http://www.atsdr.cdc.gov.

Albrecht, D. J. 2000. Sex ratio manipulation within broods of house wrens, *Troglodytes aedon. Animal Behaviour* 59(6):1227–1234.

Allan, B. B., Brant, R., Seidel, J. E., and Jarrell, J. F. 1997. Declining sex ratios in Canada. *Canadian Medical Association Journal* 156:37–41.

Anderson, R. and Bergstrom, S. 1998. Is maternal malnutrition associated with a low sex ratio at birth? *Human Biology* 70(6):1101–1106.

Astolfi P. and Zonta, L. A. 1999a. Reduced male births in major Italian cities. *Human Reproduction* 14(12):3116–3119.

Astolfi, P. and Zonta, L. A. 1999b. Sex ratio and parental age gap. *Human Biology* 71(1):135–141.

Ayeni, O. 1975. Sex ratio of live births in south–western Nigeria. *Annals of Human Biology* 2(2):137–141.

Bailey, R. C., Jenike, M. R., Ellison, P. T., Bentley, G., Harrigan, A. M., and Peacock, N. R. 1992. The ecology of birth seasonality among agriculturalists in central Africa. *Journal of Biosocial Science* 24(3):393–412.

Bandyopadhyay, M. 2003. Missing girls and son preference in rural India: looking beyond popular myth. *Health Care Women International* 24(10):910–926.

Hamilton, B. E., Martin, J. A., Sutton, P. D. 2003. Births: preliminary data for 2002. National Vital Statistics Reports Vol. 51. No. 11. Hyattsville, Maryland: National Center for Health Statistics.

Bibbins, P. E. Jr., Lipshultz, L. I., Ward, J. B. Jr., and Legator, M. S. 1988. Fluorescent body distribution in spermatozoa in the male with exclusively female offspring. *Fertility and Sterility* 49(4):670–675.

Bhat, P. N. and Zavier, A. J. 2003. Fertility decline and gender bias in northern India. *Demography* 40(4):637–657.

Bohning, D. and Ayuthya, R. S. 1999. Analysis of geographical heterogeneity in live-birth ratio in Thailand. *Journal of Epidemiology and Biostatistics* 4(2):115–122.

Bohua, L. 1986. Sex ratio at birth for urban and rural areas. *China Population Newsletter* 3(2):7–9.

Bosland, M. 1988. The etiopathogenesis of prostate cancer with special reference to environmental factors. *Advanced Cancer Research* 51:1–106.

Brewis, A., Laycock, J., and Huntsman, J. 1996. Birth non-seasonality on the Pacific equator. *Current Anthropology* 37(5):842–851.

Brown, G. R. and Silk, J. B. 2002. Reconsidering the null hypothesis: is maternal rank associated with birth sex ratios in primate groups? *Proceedings of the National Academy of Science U S A* 99(17):11252–11255.

Cagnacci A. and Volpe A. 1996. Influence of melatonin and photoperiod on animal and human reproduction. *Journal of Endocrinological Investigation* 19(6):382–411.

Cagnacci, A., Renzi, A., Arangino, S., Alessandrini, C. and Volpe, A. 2003. The male disadvantage and the seasonal rhythm of sex. *Human Reproduction* 18(4):885–887.

Cameron, E. Z. and Linklater, W. L. 2000. Individual mares bias investment in sons and daughters in relation to their condition. *Animal Behaviour* 60(3):359–367.

Carey, A. D. and Lopreato, J. 1995. The evolutionary demography of the fertility-mortality quasi-equilibrium. *Population and Development Review* 21(3):613–630.

Catalano. R. A. 2003. Sex ratios in the two Germanies: a test of the economic stress hypothesis. *Human Reproduction* 18(9):1972–1975.

Centers for Disease Control (CDC). 2003. Prostate Cancer: The Public Health Perspective: 2003 Program Fact Sheet. National Centers for Disease Control. Atlanta, Georgia.

CDC. 2000. Contribution of assisted reproductive technology and ovulation-inducing drugs to triplet and higher-order multiple births—United States, 1980–1989. *Morbidity and Mortality Weekly Report* 49(24):535–538.

Chacon-Puignau, G. C. and Jaffe, K. 1996. Sex ratio at birth deviations in modern Venezuela: the Trivers–Willard effect. *Social Biology* 43(3–4):257–270.

Chahnazarian, A. 1988. Determinants of the sex ratio at birth: review of recent literature. *Social Biology* 35(3–4):214–235.

Chambliss, R. 1949. The geographic factor in the human sex ratio at birth. *Social Forces* 28(2):190–195.

Clutton-Brock, T. H. and Iason, G. R. 1986. Sex ratio variation in mammals. *Quarterly Review of Biology* 61(3):339–374.

Clutton-Brock, T. H., Albon, S. D., and Guinness, F. E. 1984. Maternal dominance, breeding success and birth sex ratios in red deer. *Nature* 308(6735):358–360.

Clutton-Brock, T. H., Guiness, F. E. and Albon, S. 1982. *Red Deer: Behavior and Ecology of Two Sexes*. Chicago: University of Chicago Press.

Cockburn, A., Legge, S., and Double, M. C. 2002. Sex ratio in bird and mammals: can the hypotheses be disentangled? In *Sex Ratios: Concepts and Research Methods*. ed. Ian C. W. Hardy, pp. 266–286. Cambridge, England: Cambridge University Press.

Cooperstock, M. and Campbell, J. 1996. Excess males in preterm birth: interactions with gestational age, race, and multiple birth. *Obstetrics and Gynecology* 89(1):156.

Cooperstock, M., Bakewell, J., Herman, A., and Schramm, W. F. 1998. Effects of fetal sex and race on risk of very preterm birth in twins. *Obstetrics and Gynecology* 179(3 Pt 1):762–765.

Crisp, T. M., Clegg, E. D., Cooper, R. L, Wood, W. P., Anderson, D. G., Baetcke, K. P., Hoffmann, J. L., Morrow, M. S., Rodier, D. J., Schaeffer, J. E., Touart, L. W., Zeeman, M. G., and Pate, Y. M. 1998. Environmental endocrine disruption: an effects assessment and analysis. *Environmental Health Perspectives* 106(Suppl.1):11–56.

Crow, J. F. 1994. Advantages of sexual reproduction. *Developmental Genetics* 15(3):205–213.

Czeizel A. 1980. Time of fertilisation and sex of infants. *Lancet* 2(8187):199.

Dahl G., and Moretti, E. 2004. The demand for sons: evidence from divorce, fertility and shotgun marriage. National Bureau of Economic Research. Working Paper 1082. Cambridge, Massachusetts.

Darwin, C., 1871. *The Descent of Man, and Selection in Relation to Sex*. London: John Murray.

Division of Vital Statistics. 1999. *Instruction manual part 3a: Classification and coding instructions for birth records: 1999*. Hyattsville, Maryland: National Center for Health Statistics. Available on the Internet at www.cdc.gov/nchs/data/dvs/3amanual.pdf.

Davis, D. L., Gottlieb, M. B., and Stampnitzky, J. R. 1998. Reduced ratio of male to female births in several industrial countries: a sentinel health indicator? *Journal of the American Medical Association* 279(13):1018–1023.

De Jong, R. H. 1977. The pill shows no favors. *Journal of the American Medical Association* 237(14):1470.

Dickinson, H. O. and Parker, L. 1994. Do alcohol and lead change the sex ratio? *Journal of Theoretical Biology* 169(3):313–315.

Dickinson, H. O. and Parker L. 1996. Why is the sex ratio falling in England and Wales? *Journal of Epidemiology and Community Health* 50(2):114–228.

Dickinson, H. O. and Parker, L. 1997. Sex ratio in relation to fathers' occupations. *Occupational and Environmental Medicine* 54(12):868–877.

Doblhammer, G., Rodgers, J. L., and Rau, R. 2000. Seasonality of birth in nineteenth- and twentieth-century Austria. *Social Biology* 47(3/4):201–217.

Dodds, L. and Armson, B. A. 1997. Declining sex ratios in Canada. *Canadian Medical Association Journal* 156:37–41.

Egeland, G. M., Sweeney, M. H., Fingerhut, M. A., Halperin, W. E., Wille, K. K., and Schnoor, T. M. 1994. Serum 2,3,7,8-tetrachlorodibenzo-*p*-dioxin's (TCDD) effect on total serum testosterone and gonadotropins in occupationally exposed men. *American Journal of Epidemiology* 139:272–281.

Erickson, J. D. 1976. The secondary sex ratio in the United States 1969–71: association with race, parental ages, birth order, paternal education and legitimacy. *Annals of Human Genetics* 40(2):205–212.

Feitosa, M. F. and Krieger, H. 1992. Demography of the human sex ratio in some Latin American countries, 1967–1986. *Human Biology* 64(4):523–530.

Fenster, L., Katz, D. F., Wyrobek, A. J., Pieper, C., Rempel, D. M., Oman, D., and Swan, S. H. 1997. Effects of psychological stress on human semen quality. *Journal of Andrology* 18(2):194–202.

Fernandez Valades, R., Lopez de la Torre Casares, M., Castilla, J., Valladares Mendias, J. C., Ruiz Montes, A. M., Vilchez Joya, R., and Sanchez Lopez-Tello, C. 1998. Gonadal, hormonal and spermatic function in patients treated for cryptorchidism. *Cirugia pediatrica: organo oficial de la Sociedad Espanola de Cirugia Pediatrica.* 11(2):76–78.

Figa-Talamanca, I., Carbone, P., Lauria, L., Spinelli, A., and Ulizzi, L. 2003. Environmental factors and the proportion of males at birth in Italy. *Archives of Environmental Health* 58(2):119–124.

Fisch, H. and Goluboff, E. T. 1996. Geographic variations in sperm counts: a potential cause of bias in studies of semen quality. *Fertility and Sterility* 65(5):1044–1046.

Fisher, R. A. 1930. *The Genetical Theory of Natural Selection.* Oxford, England: Oxford University Press.

Fisher, R. A, 1958. *The Genetical Theory of Natural Selection.* Revised edition. Oxford, England: Oxford University Press.

Freeman, W. 1934. The weight of the endocrine glands: biometrical studies in psychiatry, No. 8. *Human Biology* 6:489–523.

Fukuda, M., Fukuda, K, Shimizu, T., Andersen, C. Y., and Byskov, A. G. 2002. Parental periconceptional smoking and male:female ratio of newborn infants. *Lancet* 359(9315):1407–1408.

Fukuda, M., Fukuda, K., Shimizu, T., and Moller, H. 1998. Decline in sex ratio at birth after Kobe earthquake. *Human Reproduction* 13(8):2321–2322.

Garenne, M. 2002. Sex ratios at birth in African populations: a review of survey data. *Human Biology* 74(6):889–900.

Garry, V. F., Harkins, M. E., Erickson, L. L., Long-Simpson, L. K., Holland, S. E., and Burroughs, B. L. 2002. Birth defects, season of conception and sex of children born to pesticide applicators living in the Red River Valley of Minnesota, USA. *Environmental Health Perspectives* 110(Suppl 3):441–449.

Gaulin, S. J., and Robbins, C. J. 1991. Trivers–Willard effect in contemporary North American society. *American Journal of Physical Anthropology* 85(1):61–69.

Geschwind, N. and Galaburda, A. M. 1987. *Cerebral Lateralization. Biological Mechanisms, Associations, and Pathology.* Cambridge, Massachusetts: MIT Press/Bradford Books.

Gibson M. A. and Mace R. 2003. Strong mothers bear more sons in rural Ethiopia. *Proceedings of the Royal Society of London. Series B. Biological Sciences* 270(Sup 1):S108–S109.

Gini, C. 1955. Sulla probabilita chextermini di una serie erratica sieno tutticrescenti (o non decrescenti) ovvero tutti decrescenti (o non crescenti) con applicazioni ai rapporti dei sessi nascite umane in intervalli successivi e alle disposizioni dei sessi nelle fratellanze umane. *Metron* 17(3–4):1–41.

Graffelman, J., Fugger, E. F., Keyvanfar, K., and Schulman, J. D. 1999. Human live birth and sperm-sex ratios compared. *Human Reproduction* 14(11):2917–2920.

Graffelman J. and Hoekstra, R. F. 2000. A statistical analysis of the effect of warfare on the human secondary sex ratio. *Human Biology* 72(3):433–445.

Grant, V. J. 1994. Maternal dominance and the conception of sons. *The British Journal of Medical Psychology* 67(Pt 4):343–351.

Grant, V. J. 1996. Sex determination and the maternal dominance hypothesis. *Human Reproduction* 11(11):2371–2375.

Graunt, J. 1662 (1975). *Natural and Political Observations Mentioned in a Following Index and Made Upon the Bills of Mortality.* (1975 Reprint Edition) New York: Arno Press.

Gray, R. H. 1991. Natural family planning and sex selection: fact or fiction? *American Journal of Obstetrics and Gynecology* 165(6 Pt 2):1982–1984.

Grech V., Savona-Ventura, C. J. Vassallo-Agius, P. 2002a. Research pointers: Unexplained differences in sex ratios at birth in Europe and North America. *British Medical Journal*. 325(7357):195.

Grech V., Vassallo-Agius, P., and Savona-Ventura, C. J. 2002b. Declining male births with increasing geographical latitude in Europe. *Journal of Epidemology and Community Health* 54(4):244–246.

Greeff, J. M. and Ferguson, J. W. 1999. Mating ecology of the nonpollinating fig wasps of *Ficus ingens*. *Animal Behaviour* 57(1):215–222.

Gu, B. and Li, Y. 1994. Sex ratio at birth and son preference in China. Paper presented at the UNFPA/KIHASA International Symposium on Issues Related to Sex Preference for Children in the Rapidly Changing Demographic Dynamics in Asia, 21–24 November, Seoul, Republic of Korea.

Guerrero, R. 1974. Association of the type and time of insemination within the menstrual cycle and the human sex ratio at birth. *New England Journal of Medicine* 291(20):1056–1059.

Guerrero, R. 1975. Type and time of insemination within the menstrual cycle and the human sex ratio at birth. *Studies in Family Planning* 6(10):367–371.

Gutierrez-Adan, A., Pintado, B., and de la Fuente, J. 2000. Demographic and behavioral determinants of the reduction of male-to-female birth ratio in Spain from 1981 to 1997. *Human Biology* 72(5):891–898.

Hama, Y., Uematsu, M., Sakurai, Y., and Kusano, S. 2001. Sex ratio in the offspring of male radiologists. *Academic Radiology* 8(5):421–424.

Hansen, A. M., Garde, A. H., Skovgaard, L. T., and Christensen, J. M. 2001. Seasonal and biological variation of urinary epinephrine, norepinephrine, and cortisol in healthy women. *Clinica Chimica Acta; International Journal of Clinical Chemistry* 309(1):25–35.

Hansen, D., Moller, H., and Jorn, O. 1999. Severe periconceptional life events and the sex ratio in offspring: follow up study based on five national registers. *British Medical Journal* 319(7209):548–549.

Henking, H. 1891. ber Spermatogenese und deren Beziehung zur Eientwicklung bei Pyrrhocoris apterus L. Zeit. wiss. *Zool.* 51.

Herman-Giddens, M. E., Slora, E. J., Wasserman, R. C., Bourdony, C. J., Bhapkar, M. V., Koch, G. G., and Hasemeier, C. M. 1996. Secondary sexual characteristics and menses in young girls seen in office practice: a study from the pediatric research in office settings network. *Pediatrics* 99(4):505–512.

Hilsenrath, R. E., Buster, J. E., Swarup, M., Carson, S. A. and Bischoff, F. Z. 1997. Effect of sexual abstinence on the proportion of X-bearing sperm as assessed by multicolor fluorescent in situ hybridization. *Fertility and Sterility* 68:510–513.

Hoyert, D. L., Smith B. L., Arias, E., Murphy S. L. 2001. Deaths: final data for 1999. National Vital Statistics Reports Vol. 49 No 8. Pub No. 2001-1120. Hyattsville, Maryland: National Center for Health Statistics.

Ingemarsson, I. 2003. Gender aspects of preterm birth. *British Journal of Obstetrics and Gynecology* 110(20):34–38.

Irving, J., Bittles, A., Peverall, J., Murch, A., and Matson, P. 1999. The ratio of X- and Y-bearing sperm in ejaculates of men with three or more children of the same sex. *Journal of Assisted Reproduction and Genetics* 16(9):493–494.

Jacobsen, R., Moller, H., Engholm, G. 1999a. Fertility rates in relation to sexes of preceding children in the family. *Human Reproduction* 14(4):1127–1130.

Jacobsen, R., Moller, J. R., and Mouritsen H. 1999b. Natural variation in the human sex ratio. *Human Reproduction* 14(12):3120–3125.

Jacobsen, R., Bostofte, E., Engholm, G., Hansen, J., Skakkebaek N. E., and Moller H. 2000. Fertility and offspring sex ratio of men who develop testicular cancer: a record linkage study. *Human Reproduction* 5(9):1958–1961.

James, W. H. 1984a. Seasonality in the sex ratio of U.S. black births. *Annals of Human Biology* 11(1):67–69.

James, W. H. 1984a. The sex ratios of black births. *Annals of Human Biology* 11(1):39–44.

James, W. H. 1985. The sex ratio of Oriental births. *Annals of Human Biology* 12(5):485–487.

James, W. H. 1986. Hormonal control of sex ratio. *Journal of Theoretic Biology* 118(4): 427–441.

James, W. H. 1987a. The human sex ratio. Part 1: a review of the literature. *Human Biology* 59(5):721–752.

James, W. H. 1987b. The human sex ratio. Part 2: a hypothesis and a program of research. *Human Biology* 59(6), 873–900.

James. W. H. 1988. Testosterone levels, handedness and sex ratio at birth. *Journal of Theoretical Biology* 133(3):261–266.

James, W. H. 1989a. Parental hormone levels and mammalian sex ratios at birth. *Journal of Theoretical Biology* 139(1):59–67.

James W. H. 1989b. Parental hormone levels and the possibility of establishing that some mammalian sex ratio variation is adaptive. *Journal of Theoretical Biology* 140(1):39–47.

James, W. H. 1990a. Seasonal variation in human births. *Journal of Biological Sciences* 22:113–119.

James, W. H. 1990b. The hypothesized hormonal control of the human sex ratio at birth—an update. *Journal of Theoretical Biology* 143(4):555–564.

James, W. H. 1992. The hypothesized hormonal control of mammalian sex ratio at birth—A second update. *Journal of Theoretical Biology* 155(1):121–128.

James, W. H. 1993. Continuing confusion. *Nature* 365(6441):8.

James, W. H. 1994. The status of the hypothesis that the human sex ratio at birth is associated with the cycle day of conception. *Social Biology* 41(3–4):278–279.

James, W. H. 1995. What stabilizes the sex ratio? *Annals of Human Genetics* 59(2):243–249.

James, W. H. 1996a. Coital rates, sex-selective infanticide, and sex ratios at birth. *Social Biology* 43(1–2):132–133.

James, W. H. 1996b. Evidence that mammalian sex ratios at birth are partially controlled by parental hormone levels at the time of conception. *Journal of Theoretical Biology* 180(4):271–286.

James, W. H. 1997a. Reproductive effects of male dioxin exposure. *Environmental Health Perspectives* 105(2):162–163.

James, W. H. 1997b. Sex ratio, coital rate, hormones and time of fertilization within the cycle. *Annals of Human Biology* 24(5):403–409.

James, W. H. 1999. Further evidence relating offspring sex ratios to parental hormone levels around the time of conception. *Journal of Theoretical Biology* 197(2):261–263.

James, W. H. 2000. Secular movements in sex ratios of adults and of births in populations during the past half-century. *Human Reproduction* 15(5):1178–83.

James, W. H. 2001. The data sources which may help strengthen the epidemiological evidence for the hormonal hypothesis of sex determination in man. *Human Reproduction* 16(6):1081–1085.

James, W. H. 2002. Smoking, sperm quality and testosterone level. *Human Reproduction* 17(12):3275–3276.

James, W. H. 2003. Sex ratios of births conceived during wartime. *Human Reproduction* 18(5):1133–1134.

James, W. H. and Rostron, J. 1985. Parental age, parity and sex ratio in births in England and Wales, 1968–77. *Journal of Biosocial Science* 17(1):47–56.

Jensen, T. K., Toppari, J., Keiding, N., and Skakkebaek, N. E. 1995. Do environmental estrogens contribute to the decline in male reproductive health? *Clinical Chemistry* 41(12 pt 2):1896–1901.

Jongbloet, P. H., Zielhuis, G. A., Groenewoud, H. M., and Pasker-De Jong, P. C. 2001. The secular trends in male:female ratio at birth in postwar industrialized countries. *Environmental Health Perspectives* 109(7):749–752.

Juntunen, K., Kirkinen, P., and Kauppila, A. 1997. The clinical outcome in pregnancies of grand grand multiparous women. *Acta Obstetricia et Gynecologica Scandinavica* 76(8):755–759.

Kalter H. 1991. Five-decade international trends in the relation of perinatal mortality and congenital malformations: stillbirth and neonatal death compared. *International Journal of Epidemiology* 20(1):173–179.

Karmaus, W., Huang, S., and Cameron, L. 2002. Parental concentration of dichlorodiphenyl dichloroethene and polychlorinated biphenyls in Michigan fish eaters and sex ratio in offspring. *Occupational and Environmental Medicine* 44(1):8–13.

Keseru, T. L., Maraz, A., and Szabo J. 1974. Letter: Oral contraception and sex ratio at birth. *Lancet* 1(7853):369.

Kiely, E. A., Chapman, R. S., Bajoria, S. K., Hollyer, J. S., and Hurley, R. 1995. Maternal serum human chorionic gonadotropin during early pregnancy resulting in boys with hypospadias or cryptorchidism. *British Journal of Urology* 76(3):389–392.

Khoury, Xi. J., Erickson, J. D., and James, L. 1984. Paternal effects on the human sex ratio at birth: evidence from interracial crosses. *American Journal of Human Genetics* 36(5):1103–1113.

Koziel, S. and Ulijaszek, S. J. 2001. Waiting for Trivers and Willard: do the rich really favor sons? *American Journal of Physical Anthropology* 115(1):71–79.

Kulch, P., Keener, S., Matsumoto, M., and Crandall, B. F. 1993. Racial differences in maternal serum human chorionic gonadotropin and unconjugated oestriol levels. *Prenatal Diagnosis* 13(3):191–195.

Lam, D. A. and Miron, J. A. 1996. The effect of temperature on human fertility. *Demography* 33:291–306.

Lazarus, J. 2002. Human sex ratios: adaptations and mechanisms, problems and prospects. In *Sex Ratios: Concepts and Research Methods*. ed. Ian C. W. Hardy, pp. 287–311. Cambridge, England: Cambridge University Press.

Lerchl, A. 1999. Sex ratios at birth and environmental temperatures. *Naturwissenschaften* 86(7):340–342.

Lin, S., Hwang, S. A., Marshall, E. G., Stone, R., and Chen, J. 1996. Fertility rates among lead workers and professional bus drivers: a comparative study. *Annals of Epidemiology* 6(3):201–208.

Lloyd, O. L., Lloyd, M. M., and Holland, Y. 1984. An unusual sex ratio of births in an industrial town with mortality problems. *British Journal of Obstetrics and Gynaecology* 91(9):901–907.

Lummaa V., Merila, J., and Kause, A. 1998. Adaptive sex ratio variation in preindustrial human (*Homo sapiens*) populations? *Proceedings of the Royal Society of London Part B. Biology* 265(1396):563–568.

Lyster, W. R. 1970. Sex ratio in the Australian capital territory. *Human Biology* 42(4):670–678.

Lyster, W. R. 1974. Altered sex ratio after London smog of 1952 and the Brisbane flood of 1965. *Journal of Obstetrics and Gynecology of the British Commonwealth* 81(8):626–631.

Manning, J. T., Anderton, R. H., and Shutt, M. 1997. Parental age gap skews child sex ratio. *Nature* 389(6649):344.

Manning, J. T., Martin S., Trivers R. L., and Soler M. 2002. 2nd to 4th digit ratio and offspring sex ratio. *Journal of Theoretical Biology* 217(1):93–95.

Marcus, M., Kiely, J., Xu, F., McGeehin, M., Jackson, R., and Sinks, T. 1998. Changing sex ratio in the United States, 1969–1995. *Fertility and Sterility* 70(2):270–273.

Martin, J. A. and Park, M. M. 1999. Trends in twin and triplet births: 1980–97. National Vital Statistics Reports Vol. 47 No 24. (PHS) 99-1120. Hyattsville, Maryland: National Center for Health Statistics.

Martin, J. A., Hamilton, B. E., Sutton, P. D., Ventura, S. J., Menacker, F., and Munson, M. L. 2003. Births: Final data for 2002. National Vital Statistics Reports Vol. 52 No. 10. (PHS) 2004-1120. Hyattsville, Maryland: National Center for Health Statistics.

Martin, J. F. 1995. Hormonal and behavioral determinants of the secondary sex ratio. *Social Biology* 42(3–4):226–238.

Martin, J. F. 1997. Length of the follicular phase, time of insemination, coital rate and the sex of offspring. *Human Reproduction* 12(3):611–616.

Martuzzi, M., Di Tanno, N. D., and Bertollini, R. 2001. Declining trends of male proportion at birth in Europe. *Archives of Environmental Health* 56(4):358–364.

Matsuda, S. and Kahyo, H. 1994. Geographical differences and time trends in the seasonality of birth in Japan. *International Journal of Epidemiology* 23(1):107–118.

Mathews, T. J. and Hamilton, B. 2002. Mean age of mother, 1970–2000. National Vital Statistics Reports Vol. 51 No. 1. (PHS) 2003-1120. Hyattsville, Maryland: National Center for Health Statistics.

McDowall, M. E. 1985. *Occupational Reproductive Epidemiology*. Office of Population, Censuses and Surveys Studies on Medical and Population Subjects, No. 50. London: Her Majesty's Stationary Office.

Mealey, L. and Mackey, W. 1990. Variation in offspring sex ratio in women of differing social status. *Ethology and Sociobiology* 11:83–95.

Melnikov, V. N. and Grech V. 2003. Seasonality of live birth sex ratio in south western Siberia, Russia, 1959–2001. *Journal of Epidemiology and Community Health* 57(6):471–472.

Menard, S. 1995. *Applied Logistic Regression Analysis*. Quantitative Applications in the Social Sciences, No. 106. Thousand Oaks, CA: Sage Publications Series.

Milham, S. Jr. 1993. Unusual sex ratio of births to carbon setter fathers. *American Journal of Industrial Medicine* 23(5):829–831.

Min, Y-I., Correa-Villasen, A., and Stewart, P. A. 1996. Parental occupational lead exposure and low birth weight. *American Journal of Industrial Medicine* 30(5):569–578.

Mocarelli, P., Brambilla, P., Gerthous, P. M., Patterson, D. G. Jr., and Needham, L. I. 1996. Change in sex ratio with exposure to dioxin. *Lancet* 348(9024):409.

Moller, H. 1996. Changes in male:female ratio among newborn infants in Denmark. *Lancet* 348(9030):828–829.

Moller, H. 1998. Trends in sex-ratio, testicular cancer and male reproductive hazards: are they connected? *Acta Pathologica, Microbiologica et Immunologica Scandinavica* 106(1):232–238.

Moynihan, J. B. and Breathnach, C. S.. 1999 Changes in male: female ratio among newborn infants in Ireland. *Acta Pathologica, Microbiologica et Immunologica Scandinavica* 107(4):365–368.

Muller, H. J. 1964. The relation of recombination to mutational advance. *Mutation Research* 106:2–9.

Myers, J. H. 1978. Sex ratio adjustment under food stress: maximization of quality or numbers of offspring? *American Naturalist* 112:381–388.

Naeye, R. L., Burt, L. S., Wright, D. L., Blanc, W. A. and Tatter, D. 1971. Neonatal mortality, the male disadvantage. *Pediatrics* 48(6):902–906.

National Center for Health Statistics. 1970. Natality public-use tape and CD-ROM, with addition of county of residence and county of occurrence data by special request to B. D. Hamilton, 2001. Hyattsville, Maryland: National Center for Health Statistics.

National Center for Health Statistics. 1980. Natality public-use tape and CD-ROM, with addition of county of residence and county of occurrence data by special request to B. D. Hamilton, 2001. Hyattsville, Maryland: National Center for Health Statistics.

National Center for Health Statistics. 1990. Natality public-use tape and CD-ROM, , with addition of county of residence and county of occurrence data by special request to B. D. Hamilton, 2001. Hyattsville, Maryland: National Center for Health Statistics.

National Center for Health Statistics. 1992. Health and demographic characteristics of twin births: 1988. Hyattsville, Maryland: National Center for Health Statistics. Available at: http://www.cdc.gov/nchs/births.htm.

National Center for Health Statistics. 2003a. Technical appendix. Vital statistics of the United States, 2002, Vol. 1. Natality. Hyattsville, Maryland: National Center for Health Statistics. Available at: http://www.cdc.gov/nchs/births.htm.

National Center for Health Statistics. 2003b. Vital statistics of the United States; 1900–1993; National Vital Statistics Reports, Part I Natality: 1994-2001. National Center for Health Statistics: Hyattsville, Maryland. Available at http://www.cdc.gov/nchs.

National Center for Health Statistics. 2003c. National Vital Statistics Reports Vol. 51 No. 4.

National Research Council. 1999. *Hormonally Active Agents in the Environment*. Committee on Life Sciences. Washington D.C.: National Academies Press.

New York Times. 2003. "India's sex ratio continues to slide." October 27, 2003.

Nicolich, M. J., Huebner, W. W., and Schnatter, A. R. 2000. Influence of parental and biological factors on the male birth fraction in the United States: an analysis of birth certificate data from 1964 through 1988. *Fertility and Sterility* 73(3):487–492.

Nonaka, K., Desjardins, B., Charbonneau, H., Legare, J., and Miura, T. 1998. Human sex ratio at birth and mother's birth season: multivariate analysis. *Human Reproduction* 13(5):1401–1402.

Norton, H. W. 1940. Note on Wood's paper on parental instinct. *Journal of Heredity* 31:29.

Nylander, P. P. 1981. The factors that influence twinning rates. *Acta Geneticae Medicae et Gemellologiae (Roma)* 30(3):189–202.

Oberg, S. 1990. Spatial mapping of sex ratios. *Popnet* 18:5–10.

Olival, A., Spira A., and Multigner, L. 2001. Contribution of environmental factors to the risk of male infertility. *Human Reproduction* 16(8):1768–1776.

Orvos, H., Kozinsky, Z., and Bartfai, G. 2001. Natural variation in the human sex ratio. *Human Reproduction* 16(4):803.

Oum, R. 2003. Beyond a strong state and docile women. *International Feminist Journal of Politics* 5(3):420–446.

Parazzini, F., La Vecchia, C., Levi, F., and Franceschi, S. 1998. Trends in male:female ratio among newborn infants in 29 countries from five continents. *Human Reproduction* 13(5):1394–1396.

Paulozzi, L. J., Erickson, J. D., and Jackson, R. J. 1997. Hypospadias trends in two U.S. surveillance systems. *Pediatrics* 100(5):831–834.

Petitti, D. B. and Wingerd, J. 1978. Use of oral contraceptives, cigarette smoking, and risk of subarachnoid haemorrhage. *Lancet* 2(8083):234–235.

Poston, D. L. Jr., Gu, B., Liu, P. P., and McDaniel T. 1997. Son preference and the sex ratio at birth in China: a provincial level analysis. *Social Biology* 44(1–2):55–76.

Potashnik, G. G., Goldsmith J., and Insler, V. 1984. Dibromochloropropane-induced reduction of the sex-ratio in man. *Andrologia* 16(3):213–218.

Reeder, A. L., Foley, G. L., Nichols, G. K., Hansen, L. G., Wikoff, B., Faeh, S., Eisold J., Wheeler, R., Warner, J, Murphy, J. E., and Beasley, B. R. 1998. Forms and prevalence of intersexuality and effects of environmental contaminants on sexuality in cricket frogs (Acris crepitans). *Environmental Health Perspectives* 106(5):261–266.

Rehan, N. E. 1982. Sex ratio of live-born Hausa infants. *British Journal of Obstetrics and Gynecology* 89(2):136–141.

Renkonen, K. O. 1970. Heterogeneity among first post-nuptial deliveries. *Annals of Human Genetics* 33(3):319–321.

Retherford, R. D. and Roy, T. K. 2003. Factors affecting sex-selective abortion in India. *National Family Health Survey Bulletin: Internal Institute for Population Studies*. 17:1.

Ridley, M. 1994. Why presidents have more sons. *New Scientist* 144(1954):28.

Roenneberg, T. and J. Aschoff. 1990a. Annual rhythm of human reproduction: I. Biology, sociology, or both? *Journal of Biological Rhythms* 5(3):195–216.

Roenneberg, T. and J. Aschoff. 1990b. Annual rhythm of human reproduction: II. Environmental correlations. *Journal of Biological Rhythms* 5(3):217–239.

Rogen, W., Beth, J., Galde, C., Yue-Liang, R., Guo, L., and Hus, C. 1999. Sex ratio after exposure to dioxin-like chemicals in Taiwan. *Lancet* 353(9148):206–207.

Rostron, J. and James, W. H. 1977. Maternal age, parity, social class and sex ratio. *Annals of Human Genetics* 41(2):205–217.

Ruder, A. 1985. Paternal-age and birth-order effect on the human secondary sex ratio. *American Journal of Human Genetics* 37(2):362–367.

Rueda-Domingo, M., Lopez Navarrete, E., Nogueras-Ocana, M., Lardelli-Claret, P., Jimenez-Moleon, J., and Zuluaga-Gomez, A. 2001. Risk factors for cryptorchidism. *Gaceta Sanitaria* 15(5):398–405.

Ryan, J. J., Amirova, Z., and Carrier, G. 2002. Sex ratios of children of Russian pesticide producers exposed to dioxin. *Environmental Health Perspectives* 110(11):699–701.

Saadat, M. and Ansari-Lari, M. 2004. Sex ratio of birth during wartime and psychological tensions. *Human Reproduction* 19(2):465.

Safe, S. H. 1996. Environmental and dietary estrogens and human health: is there a problem? *Environmental Health Perspectives* 103(4):346–351.

Sas, M. and Szollosi, J. 1980. Sex ratio of children of fathers with spermatic disorders following hormone therapy. *Orvosi hetilap* 121(46):2807–2808.

Seiver, D. A. 1985. Trend and variation in the seasonality of U.S. fertility, 1947–1976. *Demography* 22(1):89–100.

Semenza, J. C., Tolbert, P. E., Rubin, C. H., Guillette, Jr., L. J., and Jackson, R. J. 1997. Reproductive toxin and alligator abnormalities at Lake Apopka, Florida. *Environmental Health Perspectives* 105(10):1030–1032.

Sharpe, R. and Skakkebaek, N. 1996. Are oestrogens involved in falling sperm counts and disorders of the male reproductive tract? *Lancet* 341(8857):1392–1395.

Sheldon, B. C. 2004. Maternal dominance, maternal condition, and offspring sex ratio in ungulate mammals. *American Naturalist* 163(1):40–54.

Shiono, P. H., Harlap, S., and Ramcharan, S. 1982. Sex of offspring of women using oral contraceptives, rhythm, and other methods of birth control around the time of conception. *Fertility and Sterility* 37(3):367–372.

Silverman, A. Y., Stephens, S. R., Drouin, M. T., Zack, R. G., Osborne, J., and Ericsson, S. A. 2002. Female sex selection using clomiphene citrate and albumin separation of human sperm. *Human Reproduction* 17(5):1254–1256.

Singh, D. and Zambarano, R. J. 1997. Offspring sex ratio in women with android body fat distribution. Journal of Human Biology 69(4):545–556.

Soma, H., Takayama, M., Kiyokawa, T., Akaeda, T., and Tokoro, K. 1975. Serum gonadotropin levels in Japanese women. Obstetrics and Gynecology 46(3):311–312.

SPSS. 2004. SPSS 12.0. Chicago, Illinois: SPSS Inc.

Swan, S., Brazil, C., Drobnis, E. Z., Liu, F., Kruse, R. L., Hatch, M., Redmon, J. B., Wang, C., Overstreet, J. W., and The Study for Future Families Research Group. 2003. Geographic differences in semen quality of fertile U.S. males. *Environmental Health Perspectives* 111(4):414–420.

Swan, S. H., Elkin, E. P., and Fenster, L. 1997. Have sperm densities declined? A reanalysis of global trend data. *Environmental Health Perspectives* 105(11):1228–1232.

Tanaka, T. 2003. Effects of bis(2-ethylhexyl) phthalate (DEHP) on secondary sex ratio of mice in a cross-mating study. *Food and Chemical Toxicology* 41(10):1429–1432.

Tarver, J. D. and Lee, C. 1968. Sex ratio of registered live births in the United States, 1942–1963. *Demography* 5(1):374–381.

Teitelbaum, M. S. and Mantel, N. 1971. Socio-economic factors and the sex ratio at birth. *Journal of Biosocial Sciences* 3(1):23–41.

Teitelbaum, M. 1970. Factors affecting the sex ratio in large populations. *Journal of Biosocial Sciences* Supplement 2:61–71.

Toppari, J., Larsen, J. C., Christiansen, P., Giwercman, A., Grandjean, P., Guillette, L. J. Jr., Jegou, B., Jensen, T. K. , Jouannet, P., Keiding, N., Leffers, H., McLachlan, J. A., Meyer, O., Muller, J., Rajpert-De Meyts, E., Scheike, T., Sharpe, R., Sumpter, J., and Skakkebaek, N. E. 1996. Male reproductive health and environmental xenoestrogens. *Environmental Health Perspectives* 104(Suppl 4):741–803.

Ounsted, M. and Scoll, A. 1982. Social class and birthweight: a new look. *Early Human Development* 6(1):83–89.

Starfield, B. and Budetti, P. 1985. Child health status and risk factors. *Health Services Research* 19(6 pt2):817–86.

Torres-Farfan, C., Richter, H. G., Germain, A. M., Valenzuela, G. J., Campino, C., Rojas-Garcia, P., Forcelledo, M. L., Torrealba, F., and Seron-Ferre, M. 2004. Maternal melatonin selectively inhibits cortisol production in the primate fetal adrenal gland. *Journal of Physiology* 554(Pt 3):841–856.

Trivers, R. and Willard, D. 1973. Natural selection of parental ability to vary the sex ratio of offspring. *Science* 179(68):90–92.

Tuljapurkar, J. 1995. High sex ratios in China's future. *Science* 267(5199):874–876.

U.S. Bureau of the Census. 1972. County and City Data Book, 1972 [Computer file]. ICPSR ed. Ann Arbor, MI: Inter-university Consortium for Political and Social Research [producer and distributor], 1991.

U.S. Bureau of the Census. 1984a. Intercensal Estimates of the Population of Counties by Ages, Sex, and Race [United States]: 1970–1980 [Computer file]. ICPSR version. Washington, DC: U.S. Dept. of Commerce, Bureau of the Census [producer], 1984. Ann Arbor, MI: Inter-university Consortium for Political and Social Research [distributor], 2000.

U.S. Bureau of the Census. 1984b. County and City Data Book [United States], 1983 [Computer file]. ICPSR version. Washington, DC: U.S. Dept. of Commerce, Bureau of the Census [producer], 1984. Ann Arbor, MI: Inter-university Consortium for Political and Social Research [distributor], 1999.

U.S. Bureau of the Census. 1991. Geographic Mobility: March 1987 to March 1990. Current Population Reports. Population Characteristics. Series P-20, No. 456. Washington D.C.

U.S. Bureau of the Census. 1993. *We the American... Woman*. Series WE-8. Washington D.C.: U.S Government Printing Office.

U.S. Bureau of the Census. 1994. County and City Data Book, 1994 [Computer file]. Washington, DC: U.S. Dept. of Commerce, Bureau of the Census [producer], 1994.

Charlottesville, VA. University of Virginia, Geospatial and Statistical Data Center. [distributor], 2003.

U.S. Bureau of the Census. 1998. Estimates of the Population of Counties by Age, Sex, Race, and Hispanic Origin [United States]: 1990–1996 [Computer file]. ICPSR version. Washington, DC: U.S. Dept. of Commerce, Bureau of the Census [producer], 1998. Ann Arbor, MI: Inter-university Consortium for Political and Social Research [distributor], 1998.

U.S. Bureau of the Census. 2000. Women in the U.S.: A Profile. Current Population Reports. No. CENBR/00-1. Washington D.C.: Available at: http://www.census.gov/prod/2000pubs/cenbr001.pdf.

U.S. Bureau of the Census 2001. Population Profile of the United States, 1999. Current Population Reports. No. P23-205. Washington D.C.: Available at: http://www.census.gov/population/pop-profile/1999/.

U.S. Bureau of the Census. 2003a. Poverty in the United States: 2002. Current Population Reports. Consumer Income and Poverty. No. P60-22. Washington D.C.: Available at: http://www2.census.gov/prod2/popscan/P60-022.pdf.

U.S. Bureau of the Census. 2003b. *Statistical Abstract of the United States: 2002.* Washington D.C.: Available at: http://www.census.gov/prod/www/statistical-abstract-us.html.

U.S. Bureau of the Census. 2004. Geographic mobility: 2002 to 2003. Current Population Reports. No. P20-549. 2003 Annual Social and Economic Supplement. Washington D.C.: Available at: http://www.census.gov/population/www/socdemo/migrate/p20-549.html.

Uchida, H., Katayama, Y., and Nagai, M. 2000. Secular trends of sex ratios at birth in Japan. *Nippon Koshu Eisei Zasshi* 47(11):945–955.

Ulizzi, L., Astolfi, P., and Zonta, L. A. 2001. Sex ratio at reproductive age: changes over the last century in the Italian population. *Human Biology* 73(1):121–128.

Ulizzi, L. and Zonta, L. A. 1993. Sex ratio and natural selection in humans: a comparative analysis of two Caucasian populations. *Annals of Human Genetics* 57(3):211–219.

Ulizzi, L. and Zonta, L. A. 1995. Factors affecting the sex ratio in humans: multivariate analysis of the Italian population. *Human Biology* 67(1): 59–67.

Ulizzi, L. and Zonta, L. A. 2002. Sex differential patterns in perinatal deaths in Italy. *Human Biology* 74(6):879–888.

Underwood, J. H. 1993. Secondary sex ratios in Micronesian populations. *Social Biology* 40(3–4):200–206.

UNICEF. 2002. *Birth Registration: Right from the Start*. Rome: Innocenti Research Center.

United States Central Intelligence Agency (CIA). 2003. The World Factbook, 2002. Washington D.C.: CIA [producer], 2003. University of Missouri—St. Louis [distributor], 2003. Available at: http://www.umsl.edu/services/govdocs/wofact2002/.

Vartiainen, T., Kartovaara, L., and Tuomisto, J. 1999. Environmental chemicals and changes in sex ratio: analysis over 250 years in Finland. *Environmental Health Perspectives* 107(10):813–815.

Ventura, S. J., Martin, J. A., Curtin S. C., and Mathews, T. S. 1998. Report of Final Natality Statistics Vol. 46 No. 11. Hyattsville, Maryland: National Center for Health Statistics.

Ventura S. J., Mathews, T. J., Hamilton, B. E. 2001. Births to teenagers in the United States,1940–2000. National Vital Statistics Reports Vol. 49. No. 10. Pub No. 2001-1120. Hyattsville, Maryland: National Center for Health Statistics.

Visaria, P. I. 1967. Sex ratio at birth in territories with relatively complete registration. *Eugenics Quarterly* 14(2):132–142.

Weisskopf, M. G., Anderson, H. A., Hanrahan, L. P., and the Great Lakes Consortium. 2003. Decreased sex ratio following maternal exposure to polychlorinated biphenyls from contaminated Great Lakes sport-caught fish: a retrospective cohort study. *Environmental Health* 2(1):2.

Werren, J. H. and Charnov, E. L. 1978. Facultative sex ratios and population dynamics. *Nature* 272(5651):349–350.

Whiting, J. W. N. 1993. The effect of polygyny on human sex at birth. *American Anthropologist* 95(2):435–442.

Williams, F. L., Lawson, A. B, and Lloyd, O. L. 1992. Low sex ratios of births in areas at risk from air pollution from incinerators, as shown by geographical analysis and 3-dimensional mapping. *International Journal of Epidemiology* 21(2):311–319.

Wilson, K. and Hardy, I. C. W. 2002. Statistical analysis of sex ratios: an introduction. In *Sex Ratios: Concepts and Research Methods*. ed. Ian C. W. Hardy, pp. 48–92. Cambridge England: Cambridge University Press.

Winston, S. 1931. The influence of social factors upon the sex-ratio at birth. *American Journal of Sociology* 37(1):1–21.

Winters, S. J., Brufsky, A., Weissfeld, J., Trump, D. L., Dyky, M. A., and Hadeed, V. 2001. Testosterone, sex hormone-binding globulin, and body composition in young adult African American and Caucasian men. *Metabolism* 50(10):1242–1247.

Wojtowicz, M. and Jakiel, G. 2002. Melatonin and its role in human reproduction. *Ginekologia Polska* 73(12):1231–1237.

Zeitlin, J., Saurel-Cubizolles, M. J., Mouzon, J., Rivera, L., Ancel, P-Y, Blondel, B., and Kaminski, M. 2002. Fetal sex and preterm birth: are males at greater risk? *Human Reproduction* 17(10):2762–2768.

Zorn, B., Sucur, V., Stare, J., and Meden-Vrtovec, H. 2002. Decline in sex ratio at birth after 10-day war in Slovenia. *Human Reproduction* 17(12):3173–3177.

APPENDIX

Appendix A

A-1. Rectification of NCHS dataset to create common county area boundaries

County	Action
Nansemond, VA	Became independent between 1970 and 1975, merged with Suffolk City; 1970 values aggregated to Suffolk City
Washabaugh, SD	Merged with Jackson between 1970 and 1975; 1970 values aggregated to Jackson
Poquin City, VA	Independent of York County but all values recoded to York
Cibola, NM	Formed in 1981 from Valencia County; 1985–1995 values aggregated to Valencia
La Paz, AZ	Formed in 1983 from Yuma County; 1985–1995 values aggregated to Yuma County
Bronx, Richmond, Kings, Queens NY	Listed as separate counties in 1985 on NCHS data, but aggregated to New York County
Poquoson City, VA	Independent as of 1985, but all values aggregated to York County
Manassas City and Manassas Park City, VA	Independent as of 1985, but all values aggregated to Prince William Co.

VITA

Michael C. Meyers was born in 1950 in Pendleton, Oregon, to Peter and Kathleen Meyers. He graduated from Eastern Oregon University in 1971 with a B.A. in English and completed some graduate work in history and literature at Exeter College, University of Oxford, Oxford, England in 1972. He worked as a farm hand, teacher and social worker until 1978, when he began eight years of service as a regional planner and environmental manager for the Department of Interior, Bureau of Land Management in Oregon and Washington. From 1986 to 1992, he was an environmental consultant for International Technology Corp; in 1992 he co-founded Strata Environmental, Knoxville, Tennessee with four partners. Since 1998, he has been a senior GIS consultant for the University of Tennessee Institute for Public Service. He continues to research the geography of the human sex ratio at birth.